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Listerian Oration.[1]

A STUDY OF HISTORIES: A COMMENTARY ON CARDIAC PAIN OF SHORT DURATION BASED ON THE RECORDS OF FOUR HUNDRED PATIENTS.[2]

By C. H. FITTS,
Melbourne.

THERE are few people who are fortunate enough to come under the direct influence of men of the moral and intellectual stature of Lord Lister, but all of us have been influenced for the better by a schoolmaster, or by a teacher in the medical course, or by a physician or a surgeon with whom one has worked. Their fame does not often outlive their generation, but the thoughts we express to patients and students echo at times what they once said, and even our attitude of mind may have been shaped by the influence of such men. Lord Lister's early training was at University College Hospital and I have particular reason for raising from oblivion one who, when I knew him, was senior physician at that hospital and also at the Brompton Hospital, where I was his last house physician before his retirement. Dr. Batty Shaw had a phenomenal scholastic record; he passed his final examination for the Fellowship of the Royal College of Surgeons while a house surgeon at University College Hospital and won the Atkinson Morley scholarship in surgery. He was destined for the surgical staff of the hospital when he came under the spell of Dr. Sidney Ringer, and became imbued with that great physician's love of research. He then proceeded to the degree of Doctor of Medicine and to Membership of the Royal College of Physicians, and he once told me that at this point he was confronted with the choice of either a medical or surgical appointment to the staff of the hospital. I count it my good fortune that he chose to be a physician. I can see him now, in his sixty-fifth year, striding down the drive of the Brompton Hospital punctually at 4.30 o'clock each Tuesday afternoon, his attaché case designed to hold a 17 × 14 in. X-ray film, while I waited at the front door. As we went into the honorary staff room he would ask how many new patients there were, but quickly interrupted if one mentioned a diagnosis, for that took something of the pleasure from the round. He had a large consulting practice and rarely had less than 50 patients spread over the public and private wards, all of whom were the responsibility of the house physician. We would set off for the wards and gradually work our way around the hospital on both sides of the Fulham Road. We were accompanied in each ward

[1] Listerian Oration, delivered at Adelaide on April 27, 1961.

by the sister in charge and a nurse who carried a viewing box for the X-ray films. Dr. Batty Shaw took his own history and made a physical examination of the patient in the erect and recumbent positions. He then filled in his particular area of the very large history and examination sheets, complete with the diagrams of the front and back of the chest, on which he wrote his symbols; underneath he would write what he expected the X-ray film of the chest to show. I would have completed my part beforehand and underneath it was my duty to write a short commentary on the case. Then finally the X-ray film of the chest was mounted on the viewing box and we would discuss it before reading the radiologist's report. The round sometimes finished after eight o'clock at night and I have seen him walk off into the night with a volume of Plato's "Dialogues" in his case to read in the train on his journey to his home at Littlehampton. He never owned a motor-car. We would meet again at 1.30 p.m. each Saturday, when he conducted an out-patient clinic. Dr. Batty Shaw was a classical scholar who had a passion for the correct use of words which none the less fell short of pedantry. I wrote the letters to the various doctors who had referred cases, and this in itself was a liberal education, for he scanned my letters as he scanned my histories.

Perhaps enough has been said to convey something of his incomparable zest, and I cannot enlarge here on what it was that endeared him to me, for not many found it. In our discussions on medical histories, by which he set such store, and in what he made of the meeting of doctor and patient, he was unique in my experience. Perhaps I see all this in a romantic haze, and if I were to read through the histories of that year written by myself and Dr. Shaw they would appear no different from other hospital histories, and that is a chastening thought, for I believe that hospital records have a limited value. There is a variety of reasons that make them unreliable sources of information on symptomatology and physical examination for those who did not write the records, unless they are written expressly for a specific investigation. Moreover, they are cluttered up with pages of unnecessary details about investigations and with the reports of the nursing staff on diet, fluid balance and treatment. Medical records have become increasingly impersonal as they have increased in content, and senior nurses are sometimes more at home among the record sheets than among the patients. It is time that we took stock of what we wish to preserve in the hospital records department, for many of the sheets could well be destroyed, if indeed they should ever have been written, and in their room a commentary such as was beloved by Dr. Batty Shaw might find a place.

A Study of Histories.

We have reached the limit of what can be gained in clinical medicine by sense perception, and sight, touch, and hearing are unlikely to reveal anything new by physical examination. We have used for years ancillary methods such as X rays and electrocardiographs in cardiology, and the one has displayed most of what it has to offer and the other has written what it has to say. We are on the verge of a scientific world of abstraction and symbolism from which advances in knowledge will come, but in which the patient could be lost to view. I have chosen to present to you this evening a problem in which the patient, and what he has to tell, are all important. This study is based on the analysis of the histories of 400 patients in whom I believe short attacks of pain are due to heart disease. The duration of the pain has been from no longer than a moment in time up to about one hour. These observations have little statistical significance, but they are the results of a personal inquiry into the characteristics of the pain described by the patient and the subsequent history of the pain and the patient. Many of these people have had coronary occlusions and myocardial infarctions, and the reproduction of the pain in whole or in part in the major attacks has made clear the nature of the minor attacks.

Cardiac Pain of Short Duration.

The use of the term "cardiac pain of short duration" is not as a synonym for angina pectoris, though of course it embraces this. There are two defined syndromes of coronary artery disease, angina pectoris at one pole and coronary occlusion at the other; in between lies chaos and it is into this realm that I plunge. I am not dealing with physical signs, electrocardiography or radiology. I am merely taking stock of what Heberden did so supremely well when he gave "Some Account of a Disorder of the Breast" to the Royal College of Physicians, based on 20 cases, on July 21, 1768. He subsequently elaborated this in 1782, when he had seen nearly 100 people with this disorder. In the preface to his book "Commentaries on the History and Cure of Diseases" Heberden wrote: "The notes from which the following observations were collected were taken in the chamber of the sick." He went on to write that: "It appeared more advisable to give such facts only as were justified by the original papers however imperfect, than either to supply the defects from memory, except in a very few instances, or than to borrow anything from other writers." My own notes have been taken in my consulting room and they have not been specially selected. I have managed to learn the subsequent fate of most of these patients, either because they have been referred to me again, and that may be at long intervals, or because they have been problems of workers' compensation claims or because I have written either to the patient or his doctor. There is no doubtful story for which I cannot produce at least one parallel case in which the diagnosis rests on certain evidence. Short attacks of pain are usually repeated and, though the interval may be long, the opportunity to make a comparison between the first and subsequent attacks nearly always comes. Then, too, the patient is a better observer of these attacks than he is of either single or multiple coronary occlusions. For these reasons this is a richer field for the study of the vagaries of cardiac pain.

In his commentary on nearly 100 cases Heberden wrote of angina pectoris:

> They who are afflicted with it are seized while they are walking (more especially if it be uphill, and soon after eating), with a painful and most disagreeable sensation in the breast which seems as it it would extinguish life, if it were to increase or continue; but the moment they stand still, all this uneasiness vanishes.

Heberden described the pain as situated sometimes in the upper, sometimes in the middle, sometimes in the bottom of the sternum. Heberden also noted that it very frequently extends from the chest to the middle of the left arm. He went on to observe that after a year or more it will not cease so instantaneously on standing still; and it will come not only when the persons are walking, but when they are lying down, so that they are obliged to rise up out of their beds. He also wrote that it has been brought on by swallowing, coughing, going to stool, speaking or any other disturbance of the mind. "Such", he said, "is the usual appearance of the disease", but he pointed out that angina pectoris sometimes occurred while people were standing still, or sitting, or first waking out of sleep, and that it sometimes reached the right arm. Finally he described the disease in a man, aged sixty years, whose pain was entirely in the left arm for 15 years until he died suddenly. At first it came only when he was walking; after 10 years it would come upon him two or three times a week at night while he was in bed. In a few pages Heberden set down an astonishing number of facts about angina pectoris which clearly shows that he knew there was more to dolor pectoris than pain on effort. One must admire the art with which he has composed his commentary as much as one admires the acuteness of his observations. It is my purpose to fill in from my own experience some of the detail which doubtless he decided not to include.

Among my 400 patients there were few who did not at some stage describe a variation from the standard pattern of anginal pain as recognized by Heberden. It is necessary

to take a fresh history each time the patient is examined to appreciate this, for the site of the pain or its distribution, either at the beginning or during the course of the illness, might be unusual, or there might be something puzzling about the relationship of the pain to effort. In many cases a detail in the patient's narrative seemed trivial, but in another it was the important fact upon which the diagnosis depended. However unusual the symptom, I have always been able to find its mate.

Pain in the Chest.

Heberden described the pain as in the sternum, and in 83 of a group of 250 patients the pain was first felt in the chest alone. In many the pain was confined to the chest, but in 41, and doubtless in the later history of others, the pain was eventually felt elsewhere. The upper part of the sternum was most frequently indicated, often with a hand spread over the right and left sides of the chest to indicate the extent of the pain. When the pain was in the region of the xiphisternum it might almost be called abdominal, but this is confusing, and a truly abdominal onset of cardiac pain is not to be found in this group. Heberden emphasized that when the pain extends beyond the sternum there is a bias to the left. Three of my patients had pain entirely confined to the right side of the chest. In 18 cases the initial pain was at first felt outside the chest, and in some, pain in the chest never occurred.

Pain in the Arms.

When the pain was not confined to the chest it was felt in the left arm in 62 cases, in 87 in both arms and in 11 in the right arm only. This is a perfunctory statement which leaves a great deal unrecorded. Heberden said that it is uncommon for the pain to extend beyond the elbow, and I find this to be true. Indeed the commonest place in the arm indicated by the patient is the elbow, and particularly in the cubital fossa and the bony prominences. This is equally true of the pain when it is felt in both arms or in the right arm. To use the term radiation is inaccurate, for there may be no continuity between the pain in the chest and the pain in the arms. The pain may be felt only in the bony prominences of the elbows and beneath the sternum. When the pain does radiate it may spread further down one arm, commonly the left. The severity of the pain in the chest may dwarf the sensation in the arms, but the reverse may also be true and patients will describe the two areas of pain in different terms. The pain may appear upon the instant in both arms and in the chest, or one may follow the other, and one may linger when the other has vanished. "As soon as the pain reaches the arm I know it is on the way out", said a rigger climbing on high scaffolding, mistakenly believing that he had indigestion. When the pain is felt beyond the elbows its distribution may be unusual. Thus it may be confined to the flexor surface of the forearm, or the styloid process of the wrists. A man may complain that his arms feel blown up and the wristwatch is taken off. Several patients have complained of pain in the thenar eminence. The pain may radiate in conventional fashion, down the arms, and numbness and tingling are felt in the fingers. Rather than pain there may be a feeling of uselessness or heaviness. Indeed the range of sensation may vary from the agony of the arms or forearms being pulled from their sockets to something so trivial as to cause the removal of an armband from the shirt in the belief that it is too tight; yet one cannot judge the severity of the disease by the intensity of the pain.

Pain in the Throat.

By definition angina means strangling, and because this constriction was felt in the chest, Heberden wrote of dolor pectoris, and called it angina pectoris. None the less a subjective sensation in the throat, which some describe as throttling, is common. Sixty of 400 patients referred to pain or distress in the throat, generally in the cricoid region. The pain may be isolated to this area, at least for a time.

A man, aged 56 years, complained that, after playing six holes of golf, at the moment of impact with the ball he felt a throttling in the larynx. It did not happen with every shot, but by the end of the round the throttling was accompanied by pain. If he picked the ball off cleanly with a wood all was well, but if he took a divot with an iron the sensation was of considerable pain. This disquieting experience was repeated each week-end for several weeks, and became so menacing that, as summer was near, I stopped him playing. He died a violent death some months later, and at necropsy there was almost complete occlusion of the descending ramus of the left coronary artery.

Patients sometimes complain of a passing weakness or hoarseness of the voice, though not necessarily those who have pain in the throat.

Pain in the Face and Head.

There may be no distress in the throat, but pain in the region of the face and head. I have notes of 62 of 400 people who have felt pain in the mouth, pharynx, jaws, teeth or ears, and occasionally elsewhere in the head. Perhaps the jaws are most frequently affected and the patient may remove his dentures as others will remove armbands or a wristwatch.

A woman, aged 45 years, was seized with pain in both malar bones which spread down the neck into the chest to the lower sternum and through to the back. It was gone in a flash, but came again, and one day was so bad that for a few minutes she could not move. For several years she had occasional pain only in the face. Then followed six months in which she was greatly troubled by pain in the jaws. Not only did she feel the pain when moving about, but also on swallowing, and it wakened her from sleep. Electrocardiograms were normal and she was assured that her heart was not affected. For two months the pain disappeared, then unaccountably it returned, not only in the jaws but in the chest, yet a few minutes later she would be working again and lifting heavy garments without distress. There followed a coronary occlusion with very extensive anterior infarction. At no time was there pain in the arms. In another patient the pain, first felt on walking to work, in both ears, radiated thence to the chest and to the left elbow. This man died after several years while mowing the lawn, never having had an attack of pain of more than 10 minutes' duration. Last year a man, aged 44 years, died suddenly. For the preceding 12 months he had noticed severe pain in the "tonsils", particularly on towelling himself after the morning shower and on bending down. The pain had never spread beyond that area.

Central and Symmetrical Pain.

Cardiac pain may be extremely localized, and when that is so it is often central in situation, in the throat or the pharynx, the tongue or the palate, or, as in several of these patients, localized to a finger point area in or behind the sternum. It may remain localized, or from this central site it may spread widely like the ripples in a pond, or it may submerge and surface at widely separated points, sometimes in a manner so unusual that I have thought that its propagation must be along blood vessels rather than along the sensory nerves.

I have learned to look with grave suspicion on separate points of pain appearing simultaneously and symmetrically either in the thorax, the head and neck or the arms. There were 20 of 250 patients who mentioned the shoulder region particularly.

A man, aged 45 years, came into his office after lunch, sat at his desk and then felt a very unpleasant pain in the points of both shoulders. Two days later the pain came again at the same time when he was standing. It might fairly be said that these short attacks occurred after, but not during, exercise, a fact which is sometimes considered as a reason for discarding the diagnosis of angina. His third attack came a week later as he waited on a railway station; the pain persisted all the afternoon and extended down both arms. On recovery from his coronary occlusion, for the first time he began to feel pain in his chest and down the left arm.

Scatter and Shrinkage.

The pain may be widely scattered at first and later shrink. Thus a man who had long had pain on effort

and at rest, which had radiated from the centre of the chest to both jaws, the shoulder blades, the left biceps and the left thenar eminence, ultimately was restricted only by the pain in the left thumb. The pain may move against the current downwards from the head and neck, upstream from the arms to find a resting place in the chest or to travel beyond.

At the age of 51 years a woman was dressing after a shower on a hot summer afternoon; suddenly she felt a paralysing pain in the palm of the right hand and it travelled up the arm across the chest and down the left arm. It was like lightning in its passage, but unforgettable. Three uneventful years passed, till one day, when she was resting in the afternoon, again the pain came up the right arm and across the chest. It lasted about five minutes and brandy seemed to relieve her. Two days later, while sitting reading, she felt the pain in the right arm and in the region of the biceps; it was like a vice tightening relentlessly. For a long time there was no pain elsewhere and then gradually she could feel a tightening—an unusual feeling in the chest—but she emphasized that the pain was in the right arm. The electrocardiogram showed extensive anterior infarction; ventricular paroxysmal tachycardia and congestive heart failure followed, from which she made a satisfactory recovery.

Eight of 250 patients related that they had had pain in the lower limbs associated with cardiac pain in the usual areas. I am not referring to claudication, which was always sought and seldom found. Apart from this area the pain described by these 400 people was confined to the head, neck, arms and thorax, but within these confines in any patient the pain does not always have the same distribution, even in the chest.

Pain at Rest and in Sleep.

Cardiac pain of short duration may occur with effort or emotion or when the patient is seemingly at rest, or it may waken him from sleep. It is quite inaccurate to describe the angina felt when the patient is up and about as effort pain, for the truth is that the pain may occur with some trifling movement when on the same day much greater efforts are made with impunity. I have known several doctors make nonsense of exercise tolerance tests by playing vigorous tennis and squash racquets in the week-ends in the fallacious belief that the absence of pain proved that previous symptoms were not cardiac in origin. Time and again it has been made clear that there is little quantitative relationship between the pain and the exertion, and indeed effort and exertion scarcely seem appropriate terms. One hundred and sixty-two of 400 patients have had pain at rest, and this includes the so-called angina decubitus. Heberden regarded this as a later stage in the natural history of angina pectoris, and it is sometimes said that the pain is provoked by lying down in advanced cases. That is not in keeping with my experience, and White and Bland recorded angina decubitus in 20·5% of 497 cases, and found as I do that the prognosis is no worse than for angina of effort. Some patients have angina only at rest and it is of course unnerving to get unprovoked pain at unpredictable moments. Sixty of 400 patients were awakened from sleep by the pain, and the majority of these suffered from severe angina. I have repeatedly seen patients with angina decubitus demonstrate the truth of Heberden's observation, for, when seized with the pain, they will get up and stand motionless beside the bed. In the first 150 of these patients, there were 19 who had a cluster of short attacks, with or without effort. When a patient has had angina for only a few weeks and has reached the stage of having a number of attacks with or without effort in the space of one or two days, there is a grave likelihood of an occlusion taking place. All these 19 people have electrocardiographic evidence of infarction or ischæmia, and often it is difficult to be certain at what moment the occlusion began.

Intractable Angina.

There is a group of patients whose anginal pain is intractable, though the term status anginosus is scarcely applicable. I have records of 21 in 400 patients. The proportion of young people is relatively high. The pain is generally quite typical, and it occurs with great frequency often under circumstances outside the physical or emotional control of the patient, not only on exertion, but at rest and it even wakens him from sleep. The attacks are seldom of long duration; they are relieved by trinitrin and it would not be unusual for a dozen tablets to be taken in 24 hours. One man described himself as running on trinitrin like an engine on petrol. Often there are no objective signs of disease and the electrocardiogram is normal. One begins to doubt that the patient genuinely has pain and it is not difficult to find reasons to convince oneself that he has psychiatric causes for his complaint, forgetful that there are mental as well as physical components to every chronic disease. My experience is that though these people may live for years they have coronary arteriosclerosis and will eventually die as a result.

I have not long written finis to my notes on a schoolmaster afflicted with anginal pain since the age of 25 years. When I met him he was 40 years of age, and had already had the pain for 15 years, and a stellate ganglionectomy had been performed without giving him relief. His electrocardiogram was said to be normal. I was warned that he was aggressive and disillusioned, and had sought opinions both within and without the profession in great numbers. Doubt had been expressed about the basis of the pain because he complained more of the left elbow than of the chest. I knew him for the four remaining years of his life and I salute the memory of a man of singular courage who taught almost to the end of his days and never ceased to play a part in the affairs of the community in which he lived. In 20 years he never had a long attack of pain; his electrocardiogram during an attack showed great depression of the *ST* segments in the chest leads, and eventually the pattern of myocardial ischæmia. At necropsy there was complete occlusion of the anterior descending ramus of the left coronary artery at its origin, but there was no infarction. This man's grandfather died suddenly at the age of 51 years, his father at 41 years and his only brother at 34 years.

Seven of these patients with intractable pain had stellate and thoracic ganglionectomy, and only two of them gained any lasting benefit. I have not recommended the operation for a long time.

Pain on Swallowing.

Heberden wrote that some of his patients had pain on swallowing, and 18 of my 400 patients described this. In some the dysphagia was so severe that they were afraid to eat. All of such people had severe coronary disease and had had occlusions. In only one was a hiatus hernia present, and his pain was in the left arm when he was walking or swallowing cold beer. His electrocardiogram showed the pattern of myocardial ischæmia, and he had had a coronary occlusion in the past. In fairness it must be recorded that two patients claimed that drinking beer relieved the pain.

One picturesque individual earns a living well above the basic wage by selling peanuts in hotels. He works only two hours a day, during which time he visits 14 hotels. He drinks 14 beers; the first takes the pain away and "then I have no pain when the beer is in me, only when it wears out".

Another man for some time had endured attacks of pain beneath the lower part of the sternum, in both jaws, and behind the ears and eyes. He said that the pain eased when he stopped moving, but that the relief from two glasses of beer was magic; for this reason he believed that he was suffering from shiftworker's indigestion.

Length of History.

Heberden concluded that angina pectoris may continue for many years without injury to health. He gave his first paper on the subject in 1768 and he died in 1801, in his ninety-first year. According to his son he continued to see patients for many years and thus had the opportunity to prove his statement. Of 250 patients surveyed for this purpose I have known 28 for periods of nine to

20 years. A person with intractable cardiac pain would regard a day of freedom as a merciful dispensation, and a year is a long time in the life of any person with cardiac pain. I have records of 41 patients who have had at some time an interval of a year or more between attacks. There are those whose pain is related to effort who, on spending themselves less extravagantly, will lose it altogether, though they know full well that it is not far away. There are others whose pain is not related to effort and this, too, may disappear for reasons that are not obvious. I have a note of effort pain of classical distribution for a short time in 1937 and then a period of freedom until 1957.

Another patient suffered an attack of anginal pain while carrying a heavy weight of silver to the bank in 1951 and he was forced to stop until the pain ceased. Thereafter he changed his job and left the district. In 1959 I was called to see him in another part of Melbourne with coronary occlusion following a recurrence of his former pain on effort for two months.

One may wait a long time for the confirmation of a diagnosis, and confirmation and certainty may never come.

A robust friend, aged 48 years, who had spent himself in prodigal fashion for many years, complained in 1950 that for five months in his daily walk uphill to his office, and in his weekly round of golf, he felt pain high in the sternum and at the same time in both wrists. On the golf course the pain might last over four holes and he was accustomed to walking it off. He had also had the pain in bed. What is one to say of such a story when 10 years later nothing catastrophic had happened and the pain had vanished?[1]

Coronary arteriosclerosis is the foster child of silence and slow time.

Differential Diagnosis.

In a middle-aged or elderly person, in whom pain with some of the characteristics I have described is the dominant symptom, a clinical diagnosis of hiatus hernia should not be made. When the hernia is demonstrated radiologically the onus of proof still lies with those who claim that it is the cause of the pain. Many of my patients have had barium-meal X-ray examinations and 10 have proven hiatus hernia. I am prepared to believe that a routine radiological examination would disclose many more among the 400. Four of the 10 have symptoms of hiatus hernia, three of these against a background of angina of effort which has been present for many years. There is more coronary artery disease than hiatus hernia and it is a more frequent cause of pain in the chest and surrounding areas. It is common for cardiac pain to occur after meals and it is not at all unusual for patients to say that bending is one of the actions that provoke anginal pain. I have recently seen two patients in whom a diagnosis of œsophageal reflux has been made. Both had pain on effort; one had a barium-meal examination and no hernia was demonstrated; the other did not have a barium-meal examination but had ischæmic changes in the electrocardiogram; both subsequently suffered coronary occlusions. Œsophageal reflux is a well attested and important disorder, but it also tends to be a fashionable diagnosis in which proof is not always sought and may not even be found on radiological investigation.

I can speak even more strongly of the confusion in diagnosis between cardiac pain and that associated with cervical spondylosis. This has occurred twice in this series and both men had demonstrable spondylitis in the cervical spine.

One had been successfully treated on this basis for a pain in the back of the neck two years previously. On the present occasion he had been suffering short attacks of pain in the left side of the neck radiating to the left shoulder and the roof of the mouth. After two months' observation he was admitted to hospital with pain lasting one and a half hours and placed in traction. Finally, an electrocardiogram showed that he had a recent myocardial infarction. A year later he was admitted again with complete heart block and Adams-Stokes attacks.

[1] On May 19, 1961, this man died suddenly when driving his car.

An opinion frequently expressed in workers' compensation cases is that anginal pain following an occlusion is in some way brought about by the occlusion. Taking 100 of these case records there was a history of coronary occlusion with myocardial infarction in 59. Twenty-three of these people had anginal pain prior to the occlusion; 17 had the pain both before and after; 17 had it for the first time after the occlusion. Of this last group a number did not develop anginal pain for some months after the occlusion, and I cannot see that there is good evidence for this claim, which is none the less an important issue in workers' compensation.

Nitroglycerine.

What does the patient do in his extremity at the moment the pain comes? If he is moving he stands still, but he does not often sit, and often if he is lying down at the onset of the pain, he stands up. Defiant people may continue on their way until forced to stop, or, as sometimes happens, they walk it off. As they stand still they may inhale deeply, for some find this a relief, particularly when angina occurs at rest. An alkaline powder, followed by the bringing up of wind, is frequently alleged to take the pain away; but the trouble is that whatever remedy the patient employs, if he is in motion he also stands still. As with other obstacles, those who cannot go over go round, and patients learn by bitter experience to circumvent at least some of the attacks.

I have not found any long-acting nitrites of value, and they are certainly no substitute for nitroglycerine tablets, which every patient should carry. It is not too much to say that nitroglycerine is one of the therapeutic blessings of mankind. An intelligent understanding of its virtues, in both the prevention and the relief of angina, will go far to make life endurable.

I once had the care of a country doctor whose life and work bore silent witness to the fact that the greatest contribution our medical schools have made in the past has been in the provision of good general practitioners to serve the needs of widely scattered communities. He had been suffering increasingly with angina for three years and had reached a stage at which the pain was intractable. My records read like a catalogue of all reputed forms of treatment for cardiac pain, and there are odd notes and comments by the doctor himself. Here are some of them:

> If I feel a pain coming on I stand still and take a deep breath and hold it, and I feel the pain going away.
>
> The objective is to avoid or control the severity of the attacks. The more you succeed the fewer and less severe your attacks will become.
>
> The moment the attack is passed its height the tablet of nitroglycerine should be removed without delay and it can be used again. While the tablet is under the tongue keep it in motion with the tongue and swallow any saliva that forms. If the attacks are severe more tablets than one may be placed under the tongue for more rapid effect, but they must be removed as soon as possible. Up to four or five may be used in this way.

Two and a half years after sympathectomy he wrote to say that he was playing nine holes of golf, but that he occasionally required one six-hundredth of a grain of nitroglycerine. "What a wonderful drug", he wrote. You will be thinking that the doctor was a naïve person of Scottish ancestry, and you will be right.

Relief from pain after taking nitroglycerine is almost instantaneous for some, and takes a little longer for others. One patient, when asked if he was still having pains in the arms, said that now he always takes a tablet as soon as the pain comes in the chest and so prevents it reaching the arms. When I am told that it helps, but takes 10 minutes or more to take effect, I am sceptical that it is playing any part at all; indeed I doubt if the exhibition of nitroglycerine is an entirely reliable therapeutic test for the diagnosis of cardiac pain. When first given the drug, patients nearly always complain of a bursting or throbbing headache and this is understandable, but for some reason it seldom continues, even though the relief of angina is as effective as ever. It is

not that they learn to tolerate the headache, for time and again they will say that they are no longer aware of it. How long will anginal pain continue if nitroglycerine is not taken or if it fails to act? In my experience 15 to 20 minutes is not unusual, but I have many records of pain lasting up to an hour. Admittedly this is more common in those who have already had an occlusion, but it may happen repeatedly without further change in the electrocardiogram and without rise in the serum transaminase level.

Conclusion.

I have pointed out that 161 of 400 patients had pain at rest, some had it only at rest, and another 60 were wakened from sleep. All the phenomena of anginal pain may take place without a story of movement and without emotional upset. I use the term movement rather than effort to emphasize the lack of quantitative relationship between the degree of effort and the pain. There is often a periodicity about the pain, as shown by two patients who were wakened from sleep repeatedly at almost the same hour of the night, but who were free from pain by day. Yet on the other hand years may elapse between attacks and in some with the passage of time the pain may vanish. There is indeed no generalization upon which we can rely.

This study of cardiac pain of short duration suggests that there are fallacies in relating its origin too closely to effort and cardiac output. The pain is due to myocardial anoxia and is related fundamentally to considerations of coronary blood flow and oxygen consumption, and it seems clear that the biological work done by the heart is not identical with work as measured by the physicist and the engineer, nor indeed by those who expose patients to effort tests. There is no end to the ingenious explanations that can be devised to explain every attack of cardiac pain on the pathological basis of coronary arteriosclerosis, thrombosis, occlusion and myocardial ischæmia; but it may be more rewarding to seek by further study to unravel the complexities of coronary blood flow and oxygen consumption by the myocardium for, granted the presence of coronary arteriosclerosis, one cannot but be dissatisfied with the conventional idea of effort and cardiac output. I make no apology for stressing that cardiac pain is common, and I find nothing surprising in the thought that comes to me from this study that the circulation may falter once or many times in its trivial round and common task.

The subject which has been the source of my commentary presents the problem of the diagnosis of a patient's illness when there is nothing but the history upon which to base an opinion. This situation is one that I trust every medical student is made aware of and which I dare to think, and indeed to hope, will never vanish from the practice of medicine. Roger Fry said of Cezanne that:

> . . . for him the ultimate synthesis of a design was never revealed in a flash; rather he approached it with infinite precaution, stalking it, as it were now from one point of view, now from another, and always in fear lest a premature definition might deprive it of something of its total complexity.

If medicine is an art, this is its definition. It is not listening to a tale half told, whose total complexity in any case may not be visible on a single occasion. It is not awaiting the results of investigations before one begins to stalk the diagnosis, and exercising the patient with suspected angina is a poor substitute for the physician exercising his mind. It is not the stark statement that the patient has angina nor is it the explaining away of a pain with the more comforting thought of œsophageal reflux or arthritis of the spine, for in the final event it is the communication of a personal view based on many factors which may be presented to the patient by different people in different ways. Perhaps we would all agree, to borrow a word from Heberden, that this is a teasing disease both for the patient and for the doctor.

LONG-TERM ANTICOAGULANT THERAPY.[1]

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LONG-TERM anticoagulant therapy in the management of ischæmic heart disease has been a keenly debated subject since it was first proposed some 14 years ago. A physician, when faced with such a controversial topic, may well approach the problem in three carefully considered steps: (i) a critical review of the literature; (ii) an analysis of his own material; (iii) the drawing of some workable conclusion.

Review of the Literature.

In 1947 Nichol and Fassett published a preliminary report on their attempt to forestall coronary thrombosis with the aid of long-term anticoagulant therapy. This was soon followed by several papers from the United States and Scandinavia, and much later, with typical conservatism, from Great Britain. Only the more important contributions can be mentioned in this short review.

Nichol *et alii* (1958) presented to the second World Congress of Cardiology, held in Washington in September, 1954, a combined study from 10 North American clinics, reviewing 1091 patients treated since 1944. Their mortality rates for the period of observation were 6·2% for impending infarction, 10% for a single infarct and 20% for multiple infarction. They compared these rates with those of 417 so-called untreated controls submitted by some of the cooperating clinics and with those of 319 patients who for some reason abandoned treatment. The control groups, which were not strictly comparable, had much higher mortality rates; the figures for the untreated control groups were 40% for impending infarction, 37% for a single infarct and 38·1% for multiple infarction. The mortality rates for the patients who abandoned treatment were 18·7%, 18·5% and 56·1% respectively. The over-all mortality rate for the treated patients was 12%, for the untreated controls 37·4% and for the group of patients who abandoned treatment 28·2%. The total duration of therapy amounted to 2038 years and the average duration of treatment per patient was 22 months. Bleeding complications occurred in 20% of treated cases and were thought to be responsible for death in six instances (0·5% of all cases); in four of those cases death was due to cerebral hæmorrhage and in two to "subendocardial hæmorrhage".

Keyes *et alii* (1956) reported a group of 71 patients who recovered from myocardial infarction after 1947 and who were treated with long-term anticoagulants. All patients included in their series were subject to persistent post-infarction cardiac pain and thus represented a group of potentially "bad-risk" subjects. They were compared with a control group of 186 patients who recovered from myocardial infarction between 1940 and 1946. The over-all mortality rate of the treated group was 9·9%, compared with that of the control group, which was 48%. The authors state that the predicted survival time for patients with a single infarct was three times longer, and for patients with multiple infarction five times longer in the treated group than in the control series. There were 12 deaths amongst patients receiving long-term anticoagulant drugs, including three due to hæmorrhage and two due to "rebound" infarction coincidental with too sudden correction of hypoprothrombinæmia. Some bleeding complication occurred in 42% of all patients treated.

Suzman *et alii* (1955) compared 82 patients treated with long-term anticoagulants after myocardial infarction with 88 concurrently treated patients who received short-term anticoagulants only. Inclusion in the treated and untreated group depended mainly on the patient's own choice and the results are therefore not altogether comparable. How

[1] Part of a symposium on "Atherosclerosis" arranged by the Post-Graduate Committee in Medicine, University of Sydney, on July 26, 1961.

ever, there appeared to be a highly significant difference in both the recurrence rate of infarction (8·5% in the long-term group and 27·3% in the short-term group) and the over-all mortality (7·3% in the long-term group and 33% in the short-term group). The follow-up period extended for up to six years, and prothrombin indices, measured by a capillary blood method, were kept, as far as possible, between 40% and 60%.

One of the best studies is that of Bjerkelund (1957), who compared the results of patients with myocardial infarctions admitted concurrently, on an alternative basis, to two departments of the Ulleval Hospital in Oslo. In one department treatment with long-term anticoagulant therapy was practised, and in the other short-term treatment only was given. Included in the study were all patients who survived the acute attack by at least one month. Analysis of the 119 treated and the 118 control patients with regard to age, sex and severity of infarction showed that they were statistically comparable. The aim of the anticoagulant therapy was to keep the Owren prothrombin-proconvertin value in the treated group below 30%, and this aim was achieved for 82·5% of the treatment period. The mortality rate in the control group (36%) was significantly higher during the period of observation than in the treated group (20%); reinfarction occurred in 38 patients (of whom 21 died) in the control group, compared with 22 patients (of whom nine died) in the treated group. Hæmorrhagic complications occurred in 32 of the treated patients (of whom four suffered cerebral hæmorrhage) and in nine of the control patients (of whom five suffered cerebral hæmorrhage). The author cautiously concludes that his controlled clinical trial demonstrated that, correctly managed, long-term treatment with anticoagulants given by mouth, especially in patients under 60 years of age with hearts of normal size and single infarcts, resulted in a statistically significant reduction both in the incidence of recurrent infarction and in the mortality rate during the first 12 months after acute myocardial infarction. The patient material was obviously not large enough to allow Bjerkelund to come to any valid conclusions for patients over 60 years of age or for those treated for more than 12 months.

Toohey (1958) treated all patients with a severe attack of myocardial infarction and recurrent attacks cared for at New End Hospital, London, from 1955 to 1957 with a continuous régime of anticoagulants. His series includes 117 patients treated for a period of at least six months and this is compared with a control group of 327 patients treated at the same hospital between 1948 and 1955. The mortality rates, after exclusion of all non-cardiac deaths and those occurring within one month after the patient's discharge from hospital, were as follows: after six months, for the treated group 1·7%, and for the control group 11·3%; after 12 months, 11·6% and 24·3% respectively. Toohey concludes that the greatest single factor in the maintenance of adequate long-term anticoagulant cover and in the avoidance of severe hæmorrhage was experience in the regulation of anticoagulant drugs. The greater the care taken to determine the exact maintenance dose the smaller the fluctuations in the prothrombin times and hence the smaller the need for frequent blood tests.

The most important contribution to date is contained in the report to the Medical Research Council of the working party on anticoagulant therapy in coronary thrombosis under the chairmanship of Sir George Pickering (1960). A carefully controlled trial is reported, which was carried out in 15 centres in Great Britain, embracing 383 patients between the ages of 40 and 65 years who had recovered from an attack of acute myocardial infarction. By random selection these patients were placed in the treated group (and given phenindione in sufficient dosage to keep the prothrombin time between twice and 2·5 times the control values), or in the control group (and treated with a therapeutically ineffective dose of phenindione, each control tablet containing only 1 mg. of the active drug). Reinfarction occurred in 24 of the 195 treated patients during the period of observation and in 60 of the 188 controls. The mortality rates were 11% for the treated group and 17% for the control group. The report concludes that the greatest benefit from adequate anticoagulant treatment was apparent in males under the age of 55 years and that reinfarction in this group occurred only one-fifth as commonly as in the low-dosage control group. Even in patients over the age of 55 years the reinfarction rate was halved by treatment and this is regarded as statistically significant. The difference in mortality rates was most obvious in the first six months of the follow-up period, whilst the disparity in the reinfarction rate was maintained for at least two years of the follow-up period.

An important critical review of these and other series was published by McMichael and Parry (1960). They carefully analysed previously reported data dealing with the long-term prognosis of patients after myocardial infarction. The authors agree that in the acute phase of myocardial infarction anticoagulant therapy was followed by a significant reduction in the mortality rate, but in the absence of critical and controlled work they regard it difficult to define the place of anticoagulants in the prevention of the disease. All the quoted series, with the exception of that of Bjerkelund and the Medical Research Council trial, were criticized because of the lack of a comparable, random-selected control series. The only fault that could be found with Bjerkelund's trial was that his treated patients were checked at more frequent intervals than his controls. The main criticism levelled against the Medical Research Council report was that the criteria for reinfarction were not strictly defined. McMichael and Parry pointed out that in some series the control groups showed an excessively high mortality rate in the first few months after infarction, which led them to suspect that sudden cessation of anticoagulant therapy might be responsible for acute reinfarction.

One may conclude from this brief survey of the literature that the case for long-term anticoagulant therapy has not been proven to everybody's satisfaction. The perfectly controlled study designed to give an answer to the question of the prophylactic value of this form of treatment has not yet been reported, and considering that one of the series reviewed and criticized has had the backing of the Medical Research Council, the cooperation of 15 of Great Britain's major hospitals and some of the greatest experts on its working party, one must be doubtful if such a study can ever eventuate. However, it may be of significance that most of the published series, imperfect though they may be, point in one direction and report an improvement in mortality and morbidity rates in the treated patients.

The Present Series.

During the past seven years I have been associated with the long-term anticoagulant treatment of several hundred patients in hospital and consultant practice. However, the following analysis is restricted to 65 patients with ischæmic heart disease who received anticoagulant treatment for one month or longer after an attack of myocardial infarction or acute coronary insufficiency, and whose treatment was directly supervised by myself.

Of these 65 patients, 37 were continuing treatment at the time of writing, whilst treatment was suspended in 28 cases for a variety of reasons detailed below. The average duration of treatment for the whole series was 20 months; for the group continuing treatment it was 25 months; and for the group whose treatment was suspended it was 12 months. The longest period of treatment for any one patient was 76 months; 42 patients have been on continuous anticoagulant therapy for over one year, and eight patients have been treated for over three years. The total length of experience with this group amounts to 1287 treatment months or 107 treatment years. No attempt has been made to fabricate a control group.

Fifty-one of the patients were males and 14 (approximately 20%) were females. It is of some interest that the current series of 37 patients includes only three females (approximately 10%), whilst of the 28 patients whose treatment was suspended 11 were females, or nearly 40%. It appears that for some reason females are less likely to be suitable for long-term anticoagulant therapy.

The ages of the patients treated ranged from 37 to 78 years; 95% were aged between 40 and 69 years.

Forty-six patients (about 70%) suffered from myocardial infarction, of which 14 (about 20%) had a single infarction which was either unusually severe and complicated or occurred in a relatively young person, and 32 (50% of the entire series) suffered recurrent infarction which is recognized as carying a bad prognosis. Nineteen patients suffered from severe coronary insufficiency and impending infarction, with prolonged attacks of severe ischæmic pain occurring at rest, often in a sinister *crescendo* fashion.

The anticoagulant used was phenindione, which has proved a very satisfactory drug for long-term anticoagulant treatment. Toxic effects due to this drug, other than bleeding complications, although well known from the literature, were not encountered, probably because sensitization reactions usually manifest themselves in the first six weeks of treatment. One patient complained of mild diarrhœa which may have been due to drug-induced steatorrhea, but it disappeared after a few months without any specific investigations being carried out.

The aim in adjusting phenindione dosage was to keep the prothrombin time between 1·5 and 2·5 times the control time, which is equivalent to a prothrombin index of between 40% and 67%. Eighty-five *per centum* of all prothrombin index estimations were found to be within this therapeutic range. After the initial stabilization, which was usually carried out in hospital, the daily dose of phenindione, given in two nearly equal twelve-hourly doses, remained remarkably stable. If no adjustment in phenindione dosage was necessary prothrombin estimations were carried out weekly, and later at intervals of two and three weeks.

The phenindione dosage in this group of 65 patients varied from 50 to 275 mg. per day. There was a very definite trend for younger patients to require a larger maintenance dose, and the average age of the 20 patients who were stabilized on a daily dose of 175 mg. or more was 52 years. Older patients required a smaller daily dose of phenidione and the average age of the 17 patients treated with 75 mg. or less was 60 years. One of the keys to successful anticoagulant therapy is the avoidance of marked fluctuations in prothrombin levels, and for this reason any alteration in the daily phenindione dosage, after initial stabilization, rarely exceeded 25 mg.

It is of interest to study in some detail the reasons for the suspension of treatment in 28 cases. In six cases the suspension was due to the death of the patient; four of these deaths were sudden, one was due to pulmonary œdema and one was due to cerebral hæmorrhage. In eight cases bleeding occurred; this was from the gastro-intestinal tract in two cases and in two instances from hæmorrhoids (one of these patients recommenced anticoagulant therapy after surgical treatment); hæmaturia occurred in three cases (one patient recommenced anticoagulant therapy later) and menorrhagia in one case. The suspension was due to intercurrent hepatitis in one case, and to geography in four cases, three of the patients continuing or recommencing therapy elsewhere. Nine patients were suspended at their own request; three of these recommenced because of the return of symptoms, one died from a myocardial infarction and five remained well without anticoagulant therapy.

Six patients died whilst on anticoagulant therapy. Four male patients between the ages of 44 and 61 years died quite suddenly and may well have suffered from a recurrence of acute myocardial infarction, but unfortunately no autopsy confirmation is available. All had had two or more infarcts before commencing long-term anticoagulant treatment, one was a moderately severe diabetic as well and another suffered from advanced hypertensive cardiovascular disease, but appeared to be well controlled with ganglion-blocking drugs. They had been receiving treatment for periods varying from seven to 15 months prior to their fatal attack. It may be relevant that the last prothrombin index in three of these four cases was 65% —a figure which, although accepted in this paper as being within the therapeutic range, may yet prove too high for special risk patients, such as relatively young males with recurrent myocardial infarction. The fourth patient, the diabetic aged 61 years, had a prothrombin index of 45% a few days before his death.

The fifth patient, a female, aged 51 years with recurrent myocardial infarction, died in congestive heart failure with pulmonary œdema not long after commencing long-term anticoagulant treatment. The sixth fatality occurred in 1956 and may well have been due to faulty case selection and faulty technique in treatment. The patient was a severely hypertensive female, aged 58 years, who was subject to repeated and disabling attacks of acute coronary insufficiency. Her prothrombin levels were kept too low and a few days before her fatal cerebral hæmorrhage her prothrombin index was 26%. Nowadays such a patient would certainly be managed more conservatively, and if she was regarded suitable for long-term anticoagulant therapy her prothrombin levels would certainly be kept higher. However, it is somewhat comforting to recall that in Bjerkelund's series five patients in the control group died of cerebral hæmorrhage as against four in the treated group.

Eight patients abandoned treatment on account of bleeding complications. Two patients with gastro-intestinal hæmorrhage required blood transfusions, and in one of them a previously unsuspected peptic ulcer was discovered. None of the other bleeding complications required transfusion or vitamin K_1 therapy—all that was necessary to stop the hæmorrhage was the withdrawal of the anticoagulant. Two patients had to stop anticoagulant treatment on account of rectal bleeding; both came to hæmorrhoidectomy and one of them subsequently recommenced treatment. A third patient in this series had a hæmorrhoidectomy; in this case anticoagulant therapy was interrupted for a few days only for surgical treatment. Three patients stopped treatment after brisk hæmaturia, and one of them subsequently resumed treatment on account of severe cardiac symptoms. One female, aged 37 years, the youngest patient in this group, developed severe menorrhagia and after several unsuccessful attempts had been made to lower the phenindione dosage during menstruation, treatment was abandoned. Similar difficulties have been previously reported.

One patient, after continuous treatment for two and a half years, developed a severe attack of hepatitis, probably of viral origin during a mild epidemic, and had to discontinue therapy.

Four patients were removed from the currently treated series for geographical reasons—that is, an overseas trip or transfer to the care of another medical practitioner. Three of these four patients have either recommenced anticoagulant therapy or are continuing under somebody else's supervision. In nine cases the treatment was stopped at the patient's own request, either because the regular blood tests were becoming tiresome, or because the absence of symptoms made it hard for them to appreciate the desirability of close medical supervision. One patient died from myocardial infarction 18 months after the cessation of treatment, three patients subsequently recommenced anticoagulant treatment on account of threatening cardiac pain, and five patients are continuing to do well without anticoagulant treatment, two of them as long as five years after the suspension of therapy.

Although bleeding complications were common in this series only three major hæmorrhages occurred amongst the 65 cases in the series — not an excessive incidence in view of the fact that treatment was continued in some cases for several years, and the total duration of anticoagulant cover extended over 1287 treatment months. The three major bleeding complications have already been discussed—namely, the case of cerebral hæmorrhage and the two cases of gastro-intestinal hæmorrhage. Minor bleeding complications included hæmaturia (15 cases), hæmorrhoidal bleeding (seven cases), severe bruising (four cases), bleeding gums (three cases), hæmatoma

formation (three cases), hæmoptysis (three cases), epistaxis (three cases), bleeding at dental extraction (two cases) and menorrhagia (one case). Some bleeding complication occurred in half the patients treated, but only three patients suffered serious hæmorrhage.

Hæmaturia was the commonest form of bleeding and occurred in one-fifth of all the patients treated. All patients were warned to look out for this complication and during the past two years have been issued with "Occultest" tablets as a routine to enable them to detect minor degrees of hæmaturia. In most cases the temporary suspension of the anticoagulant drug was sufficient to stop the bleeding, and patients were instructed to drink plenty of water to avoid clot colic. In three cases this complication was serious enough to lead to the termination of anticoagulant therapy. Hæmorrhoidal bleeding was the second commonest bleeding complication and necessitated hæmorrhoidectomy in three cases. Bleeding after tooth extraction occurred in two cases early in this series; nowadays patients are warned about this possibility as a routine.

Other minor bleeding complications were observed which responded readily to a transient reduction in phenindione dosage or the temporary suspension of treatment. Difficulties with these would arise only in the case of unintelligent and uncooperative patients failing to take notice of obvious warning signals; however, such patients have been considered unsuitable for this form of treatment right from the very beginning.

It is of importance to stress that even in this selected group bleeding manifestations were observed in 33 of the 65 patients. Close and continuous supervision is therefore one of the absolute prerequisites of this form of therapy.

Discussion.

After reviewing some of the important contributions to the extensive literature on long-term anticoagulant therapy and analysing the personal experience gained during the past seven years one may come to certain tentative conclusions. Long-term anticoagulant therapy may be considered for the following groups of patients: (i) those with recurrent myocardial infarction and possibly a single infarct, especially in the presence of complications or in a young person; (ii) those with acute coronary insufficiency with repeated attacks of severe ischæmic rest pain (the so-called "impending myocardial infarction"); (iii) those with rheumatic heart disease with atrial fibrillation and recurrent emboli; (iv) those with certain forms of cerebrovascular disease, such as intermittent insufficiency of the basilar arterial system or intermittent insufficiency of the carotid arterial system; (v) those with recurrent and life-threatening thromboembolic disease of varying ætiology.

Absolute contraindications to long-term anticoagulant therapy include: (i) bleeding disorders; (ii) active peptic ulceration; (iii) hepatic insufficiency; (iv) advanced renal disease. Great caution is required during dental extractions and minor surgery, in females during menstrual periods, in patients with severe hypertension and in all elderly patients, arbitrarily defined as being over the age of 70 years. Patients must be warned concerning the use of salicylates, phenylbutazone and broad-spectrum antibiotics, which greatly potentiate anticoagulant action.

Three basic essentials for successful long-term anticoagulant therapy are: (i) an intelligent patient, who is prepared to cooperate in this form of treatment, fully aware of the risks of bleeding complications and trained to recognize them early; (ii) an enthusiastic physician, who is willing to accept responsibility and to supervise personally all aspects of treatment and ready to give meticulous attention to all details; (iii) a reliable laboratory, which is prepared to check its method of prothrombin estimation frequently, always comparing its results with several controls.

It is virtually impossible for any single person to arrive at a dogmatic conclusion as to the prophylactic value of long-term anticoagulant therapy. One could quote only a large number of individual case records, which are suggestive but certainly not convincing evidence as to the value of this form of treatment; one has often regretted not having placed a patient on long-term anticoagulant drug treatment. It is sufficient for the purpose of this communication to state that from the practical point of view this is a feasible form of therapy, and that the risk incurred is not unreasonable in view of the prognosis of the majority of the patients subjected to treatment.

Summary.

Long-term anticoagulant therapy in the management of ischæmic heart disease continues to be a keenly debated and highly controversial subject. Some of the most important contributions to the literature have been reviewed. The results in a personal series of 65 patients treated for up to 76 months have been critically analysed. Whilst no dogmatic conclusions as to its prophylactic value may be warranted, experience has shown that, provided certain fundamental rules are observed, long-term anticoagulant therapy is practicable and that the risk incurred is not unreasonable in view of the prognosis of patients subjected to treatment.

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SEROLOGICAL EPIDEMIOLOGY OF ARBOR VIRUSES IN WESTERN AUSTRALIA.

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THESE preliminary investigations of the epidemiology of arbor viruses in Western Australia were stimulated firstly by the fact that no extensive serological examination for arbor viruses had been carried out in the western half of this continent, and that it was therefore considered desirable to obtain serological data that could be compared with other areas of Australia; and secondly by the observations of Shope and Anderson (1960) of the possible association of a group A arbor virus infection of man with the clinical syndrome of so-called epidemic polyarthritis.

In this regard Anderson *et alii* (1961) have demonstrated that a useful indicator of group A antibody was the virus known as AMM 2354 (isolated from culicine mosquitoes by Dr. E. Buescher in Malaya). In 1958, Dr. D. J. R. Snow,

TABLE I.

Results of Hæmagglutination-Inhibition and Neutralization[1] Tests with MVE and AMM 2354 Arbor Viruses on 673 Specimens of Human Serum.[2]

Sera.	Locality.	Percentage of Sera with Antibodies Demonstrable by			
		Hæmagglutination-Inhibition Tests.		Neutralization Tests.	
		MVE (B)	AMM 2354 (A)	MVE (B)	AMM 2354 (A)
White (207)	South-west (1958).	34	28	3	10[3]
White (50)	Kimberley (1960).	60	25	18	4
Aboriginal (41)	Central Australia (1958)	0	100	0	45
Aboriginal (285)	Kimberley (1960).	92[4]	50	(50)	(68)
Aboriginal (86)	Leonora (1958).	4	22	0	28
Aboriginal (25)	Carnarvon (1958).	17	8	11	8

[1] Only 420 sera were tested for neutralizing antibody.

[2] Positive sera are recorded as those giving positive results in titres of 1/10 or more for neutralization tests and in titres of more than 1/10 for hæmagglutination-inhibition tests.

[3] In the Busselton area this rose as high as 38%.

[4] 30% gave positive results in titres greater than 1/320.

of the Department of Public Health, Western Australia, described an outbreak of polyarthritis with rash occurring in 1956 in the Busselton area in the south-western part of Western Australia—an area which, according to Anderson, was apparently not infected with MVE virus in 1951 (Anderson *et alii*, 1952). Some epidemiological findings (Anderson and French, 1957) have suggested that the natural histories of the viruses of MVE and epidemic polyarthritis may be similar, but more recent evidence conflicts with this view (Anderson *et alii*, 1961). For this and other reasons, representatives of both groups A and B were used in the attempt to obtain an antibody pattern.

Materials and Methods.

Through the courtesy of Dr. Anderson we received the AMM 2354 strain and used this as a representative of group A. A strain of MVE was selected as a representative of group B. Neutralization and hæmagglutination-inhibition serological tests were made to detect antibody against these agents.

The source of hæmagglutinin was the brains of infected suckling mice and the method of preparation was a modification of that of Casals and Brown (1954) which employed the sucrose acetone-extraction method. Sera were treated with kaolin and then with washed goose erythrocytes before the hæmagglutination-inhibition test was performed. Serial double dilutions of serum from 1/10 to 1/640 were used. In all tests goose red cells were employed.

Neutralization tests were carried out in infant mice; the intraperitoneal route of inoculation was used for serum-virus mixtures as employed by Stanley *et alii* (1951) for Coxsackie viruses. One hundred ID_{50} of virus were employed and the serum dilutions were initially tested at a dilution of 1/10. Sera showing protection were then titrated to a final dilution of 1/640.

The sera were collected from aborigines, white people, birds and animals from different parts of the State. In this preliminary survey 780 sera were examined. The sera were maintained at –20° C. until examined.

Results.

Antibody in Human Sera.

The results of hæmagglutination-inhibition and neutralization tests performed on 673 specimens of human sera are summarized in Table I. The numbers in the four columns on the right refer to the percentage of sera with antibodies. The numbers in the column under the heading "Sera" refer to the total number examined in any one locality from either white people or aborigines. The figures in parentheses for the aboriginal native sera in the Kimberley district refer only to the 92% and 50% of sera showing positive results to hæmagglutination-inhibition tests and not to the total number of 285. Hall's Creek and Derby (Figure I) were the main collecting centres in the Kimberley area. In the south-western area, where the aboriginal population is relatively small, collections of white sera were made over a wide area. It will be noted that only 10% of 207 sera gave protection to mice in the neutralization test. An analysis of the results showed that there was a higher percentage (38%) of positive results in the Busselton area than elsewhere.

Antibody in Sera Other than Human.

Results of hæmagglutination-inhibition tests on animal sera are recorded in Table II. Owing to the small numbers of sera tested these are not given as percentages and only the results of the hæmagglutination-inhibition test are presented. It will be noted that neutralization tests on the two positive kangaroo sera gave negative results in the case of MVE and that only two horse sera gave positive results with AMM 2354. It is also pertinent that all fowl sera tested gave negative results with AMM 2354.

TABLE II.

Results of Hæmagglutination-Inhibition Tests with Animal Sera.

Sera.	Locality.	Number Tested.	Number with Titres Greater than 1/10.	
			MVE.	AMM 2354.
Fowl.	Perth.	33	2	0
Kangaroo.	Leonora.	16	2[1]	0
Quokka.	Rottnest.	18	0	0
Horse.	South-west.	8	2	2
Cow.	South-west.	21	6	0
Pig.	South-west.	11	0	0

[1] Neutralization tests gave negative results.

Antibody to AMM 2354 in the Sera of Patients with Polyarthritis and Rash.

Table III gives the results of neutralization tests on the sera of patients believed to have developed polyarthritis at the time of the 1956 outbreak in the south-western part of the State as described by Snow (1958). It will be seen that only four of 16 sera gave protection. The titres varied between 1/80 and 1/320. Of two patients developing this syndrome in the metropolitan area one had serum which gave a positive result (titre 1/320) and the other had no detectable antibody.

Attempts to Isolate Virus from Mosquitoes.

Approximately 4400 mosquitoes, comprising species of the genera *Aëdes*, *Culex* and *Anopheles*, were collected in the south-western area of the State during 1960. Virus was not isolated from any of the batches constituting this pool by intracerebral and intraperitoneal inoculation of infant mice. Mosquito collections for virus isolation have not yet been made in the north-western area.

Discussion.

There is an obvious difference in the distribution of group A and group B antibody in Western Australia. This may be clearly seen in the map (Figure I), where the results given in Table I have been placed in the geographical environment.

TABLE III.
Results of Neutralization Tests with AMM 2354 on Convalescent Sera.

Time After Onset.	Number Tested.	Number Positive.
4 years	16	4
3 weeks ..	2	1

Some epidemiological findings of other workers support the view that the natural histories of the viruses of MVE and epidemic polyarthritis are similar, but Anderson *et alii* (1961) point out that there are some differences of distribution as well as the absence of avian antibody to AMM 2354. Not only have we confirmed the absence of group A avian antibody, but the Western Australian pattern emphasizes the difference of the group A and group B viruses studied. Group B antibody occurs in very

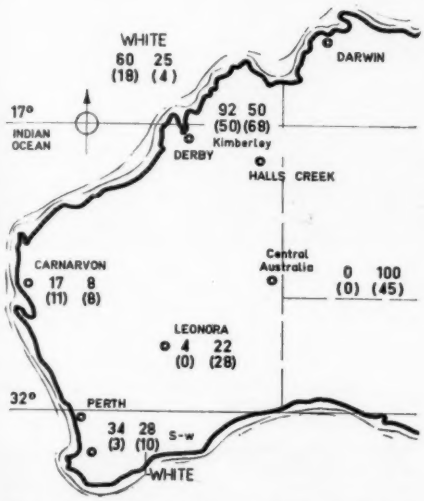

FIGURE I: Map showing location of centres from which sera were collected. The numbers refer to the percentages of sera showing positive results to tests for the presence of antibody (see also Table I). The numbers on the top refer to the results of hæmagglutination-inhibition tests and the lower numbers in parentheses to the results of neutralization tests. The numbers on the right refer to the results with AMM 2354 (group A) and those on the left to MVE (group B). Unless indicated by the word "white" all percentages refer to the sera taken from aborigines.

high incidence in the tropical Kimberleys, it is of considerably lower incidence in the Carnarvon district and it is almost non-existent elsewhere.

In Central Australia (Rawlinson Range) and the Leonora-Laverton district group A antibody only was detected. These areas should be particularly suitable for the investigation of possible vertebrate hosts and mosquitoes for AMM 2354 antibody or virus. Although all native sera from Central Australia had hæmagglutination-inhibition antibody to AMM 2354, only 45% possessed detectable neutralizing antibody. This difference could be explained by the assumption that either more than one group A virus has been in the Rawlinson Range area or that AMM 2354 is closely related to, but not identical with, the agent causing epidemic polyarthritis in Western Australia. In the Leonora-Laverton district this difference was not observed.

In the Kimberley district the percentage of sera containing MVE neutralizing antibody was considerably lower than the percentage with hæmagglutination-inhibition antibody against MVE with sera collected from both black and white populations. These results suggest the presence of more than one group B arbor virus in this area. The observation is of particular interest in view of the isolation of hitherto undescribed group B viruses in northern Queensland by Doherty *et alii* (1961). In addition to dengue and Japanese B, Doherty's MRM 16 and MRM 32 prototype strains could well be used in any attempt to elucidate the problem in the north-western part of Australia.

In the Kimberley area, too, the percentage of native sera with hæmagglutination-inhibition and neutralizing antibody to both group A and group B viruses was significantly higher than in the white population. It is not known what factors are responsible for this difference, although several may be involved—for example, mobility, socio-economic considerations (including vector-control), location with respect to rivers and so on.

Sera were not collected from children under 11 years of age and there appeared to be no significant distribution of antibody with respect to age.

The negative results attending attempts to isolate virus from mosquitoes suggest that "sentinel" suckling mice should be used in addition to the classical methods employed.

Summary and Conclusions.

Group B antibody has a high incidence in the north and a somewhat lower incidence in the north-west, and is almost non-existent elsewhere in Western Australia. Group A antibody is found over the entire State in both black and white populations. Group A antibody has not been detected in avian sera. Results of neutralization and hæmagglutination-inhibition tests suggest the presence of group B viruses in addition to the virus of MVE in the Kimberley district. Further serological and virus-isolation studies are required to explain the findings presented.

Acknowledgements.

The authors wish to express their thanks to Dr. D. J. R. Snow, Dr. R. L. Kirk and Dr. W. R. Pitney for the collection of sera; to Dr. D. J. R. Snow and Mr. E. J. Britten for the collection of mosquitoes; to Dr. S. G. Anderson and Dr. R. L. Doherty for supplying viruses; and to Miss R. Thambiah for carrying out some of the hæmagglutination-inhibition tests.

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THE FAILURE OF STAINLESS STEEL IN THE TREATMENT OF FRACTURES.

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THE frequency with which metallic implants, particularly those of stainless steel, are used for fixation in traumatic surgery speaks highly of their strength and efficiency. However, two recent disasters have befallen us in this field, the first during the use of a fixed internal splint and the second in the use of a sliding internal splint. These will be discussed separately.

Reports of Cases.

CASE I.—A boy, aged 19 years, was admitted to hospital with a transverse fracture of the left femur in its middle third, which was treated by means of a Küntscher nail. The limb was immobilized in a resting Thomas splint for two weeks. Active quadriceps drill was begun after seven days. He was allowed to walk with crutches after three weeks and returned to weight-bearing and activity at seven weeks. Nineteen weeks after the accident he was walking along the road when he felt pain at the site of injury and could not proceed further. X-ray examination showed that a fracture had occurred in the nail at the site of fracture of the femur. The patient was immediately readmitted to hospital and a new nail and bone graft were inserted. He was able to return to work five months after the second operation, the fracture having consolidated in the meantime.

CASE II.—A woman, aged 68 years, was admitted to hospital four hours after sustaining a transcervical fracture of the left femur. The fracture was treated by means of a Charnley lag screw with compression. The wound healed satisfactorily. She was allowed out of bed 24 hours after the operation. Mobilization was begun 10 days after the injury, when the sutures were removed. Immediately the patient began to complain of pain whenever she put any weight on the injured leg, though no fever occurred. This pain increased, despite antibiotic therapy on the one hand and analgesic administration on the other, so that six weeks after its insertion the apparatus was removed. It produced no growth on culture, and the pain ceased immediately.

Investigation.

Examination of the Küntscher nail showed that at the site of fracture there was a pit through which the fracture had occurred (Figure I). When this was examined under reflected light at 20 diameters of magnification it was observed that the longitudinal polishing scratches of the nail extended across the edges of the defect. It was clear therefore that this pit had occurred during manufacture—a fact which would probably not have been of any consequence if it had not happened to lie at the site of the original fracture. The actual hardness was estimated at various points along the nail and it was found to vary from about 15 Rockwell C units to 37 Rockwell C units at the site of the fracture, confirming that a high degree of work hardening had taken place at this point. The presence of the pit had thus led to a concentration of stress with work hardening under strain and fatigue fracture.

Examination of the compression screw after removal showed that an area of pitting 5 mm. by 5 mm. existed on the shank of the screw and inside the sleeve of the plate through which it passed. The area of roughening was on the superior aspect of the screw and the area of the plate in contact with it. The medial margin of the sleeve appeared to be intact, there being no evidence of wearing in this situation. The medial aspect of the pitted area on the screw was almost a straight line and was identical with the margin in the opposing element. It would appear therefore that an area of electrolytic activity had developed in this area of maximal movement of the two parts of the apparatus. It could be argued that this is the area of maximal wear, but if wear were responsible for the abnormal surfaces it would be most obvious at the edge of the pit. Electrolytic activity at this site is therefore the only satisfactory explanation for the pitting.

Commentary.

In the consideration of any implant, two conditions outweigh all others. First, the implant should be completely

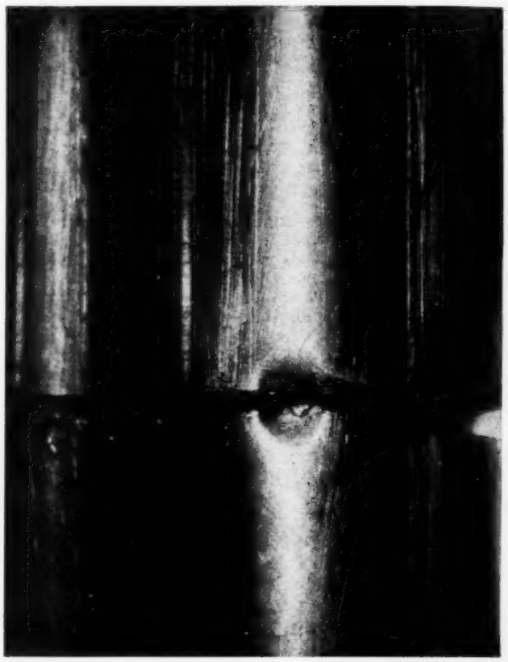

FIGURE I: The fracture line in the Küntscher nail showing the pit at the site of fracture and the longitudinal scratch marks acquired during polishing of the nail.

reliable for the purpose for which it is designed, and secondly, it should be inert in the tissues. There are many reasons for failure of a splint, two of which are

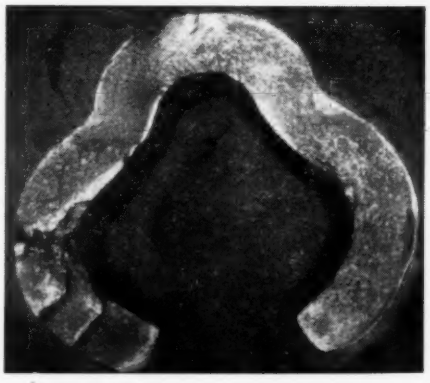

FIGURE II: Photograph of the fractured Küntscher nail showing the pit in the nail on the left-hand side, with the irregularity in the immediate vicinity (× 6).

illustrated in these two case reports. In the first case, the Küntscher nail failed because of weakness and hardening at the site of fracture. The physical charac-

teristics of the second implant were such that although it was mechanically efficient, one area of the implant failed and gave rise to considerable electrolytic activity and a sterile inflammatory response in the region of the greater trochanter.

The alloys of which each of these splints were made belonged to the group of 18-8 Austenitic steel (18% chromium and 8% nickel, with varying quantities of other substances to about 3% or 5%, the balance being

FIGURE III: The area of pitting on the shank of the lag screw. The linear nature of the medial edge of the erosion is very obvious (× 1·5).

iron). These steels are nominally non-magnetic in the normal annealed and passive state. However, after work hardening the magnetic properties may change and both of the implants discussed were magnetic when tested. The lag screw in the Charnley splint was brightly polished apart from the one pitted area. After the collar of this apparatus had been bisected it was found that the inner aspect was quite rough, carrying the drill turning marks from the time of its original fabrication. No effort

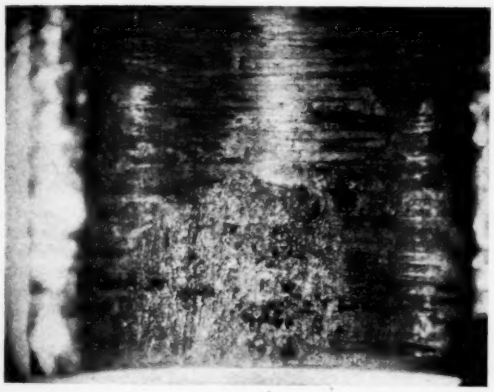

FIGURE IV: Photograph of the inner aspect of the sleeve corresponding in position to the area on the screw. It will be seen that the circumferential cutting is very obvious except in the area of erosion where grain structure has been exposed. In this area at the medial margin of the specimen there is a change in direction of the fissuring so that it is longitudinal. This would indicate that there was movement in this direction during compression and in the life of the apparatus (× 7·5).

whatsoever had been made to polish this aspect. It would appear therefore that a fragment of some foreign metal had lodged at the site of activity, or alternatively that an area of abnormal composition had been opened up by the drilling, allowing electrolytic activity to develop at this site.

The loss of the passive layer, normally produced by polishing and treatment with nitric acid to produce a film of oxide on the surface of the stainless steel, allowed corrosion to progress. This is the most serious drawback to the use of stainless steel, for if the passive layer is lost the material behaves like a simple ferrous alloy and becomes tarnished.

The cellular response on the part of the host to the compression screw was one of mild inflammation. There was round-cell infiltration in the soft tissues and in the bone. Immediately after the implant was removed the symptoms abated.

Summary and Conclusions.

1. The reasons for failure of two commonly used internal splints is discussed.

2. In the case of the Küntscher nail the failure was due to fatigue fracture in the line of a fault in the nail.

3. In the compression screw an area of electrolysis was discovered which accounted for pain in the area before removal of the foreign body.

4. The loss of the passive surface from stainless steel leaves a base metal area which will corrode rapidly unless the passive area can be reconstituted quickly. This usually occurs if the surface is polished and smooth, but when the surface is rough it is less likely that the passive state will be reconstituted.

A NEW APPROACH TO THE OPERATION OF POSTERIOR COLPO-PERINEORRHAPHY.

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THE anatomy of the perineal region and the pelvic floor should be well known; it is quite adequately described in most textbooks of anatomy. It is all the more surprising, therefore, that one of the commonest surgical procedures—namely, posterior colpo-perineorrhaphy—which is a reparative procedure and therefore should be strictly anatomical, has been performed for the past 50 years in more or less the same way, with little regard for anatomical principles.

Fallacies.

Many surgeons who perform this operation fail to realize that the levatores ani muscles do not form parallel bands of tissue immediately deep to the perineal skin, but are arranged in the form of an inverted cone (Figure I), the broad base of which is uppermost and arises from about the middle of the cylinder-like walls of the pelvis, with the apex directed downwards and inserted into the muscle coats of the ano-rectal canal and related structures above the anal sphincter, about one and a half inches from the anal orifice. By far the strongest portion of this muscle lies anteriorly and consists of the pars pubo-rectalis, which is half an inch or more in thickness, and surrounds and is attached to the vaginal walls about two inches above the vaginal introitus. Its fibres sweep downwards and backwards on either side in an arcuate manner concave downwards from about the middle of the pubic bones to reach their insertion at the ano-rectal junction.

So much interlacing of the fibro-muscular tissue takes place in this region that in the repair of the perineal body it is easy to pick up a broad band of solid-looking tissue anywhere from the perineal skin to a point two inches above it and, by the exercise of downward traction on the anal sphincter, this band can be made to resemble (indeed, it is usually mistaken for) the medial borders of the partes pubo-rectales of the pubo-coccygeal portions of the levatores ani.

The idea that in order to form a solid pelvic floor it is necessary to unite the levatores ani for a distance of one and a half to two inches in front of the anus is quite wrong. These muscles, except for their junction in the ano-rectal ring at the apex of the perineal body, were never intended by nature to be united. If the surgeon is successful in uniting them in this artificial and arbitrary manner, ample evidence has been supplied by means of radio-opaque markers and other means to show that they

remain in apposition for a few weeks only, and that in accordance with surgical principles their tendency to separate is inevitable.

If the partes pubo-rectales are not united at the point where nature intended them to be united—at the apex of the perineal body—but instead the fibro-muscular tissues at the base of the perineal body are united, then eventually a band of weak scar tissue is formed, clothed on either side by modified skin. This situation is quite unsatisfactory, tending inevitably towards a recurrence of prolapse or the development of fresh prolapse.

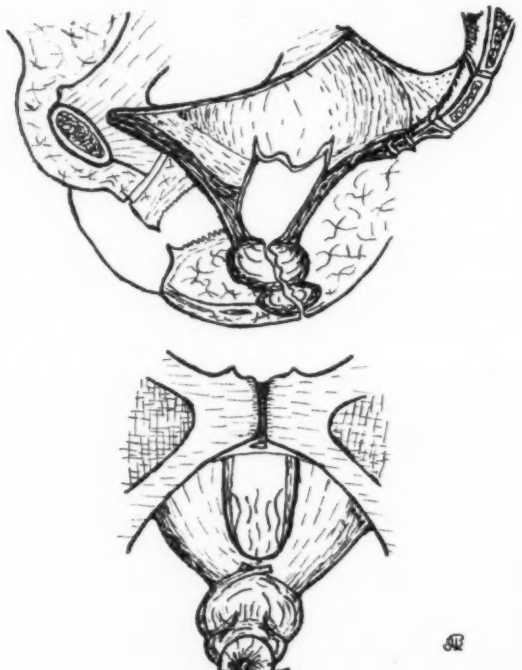

FIGURE I.

Twenty-seven years of surgical practice, of which the last 15 were spent in a large gynæcological clinic, have shown that the results of surgical treatment of prolapse are far from satisfactory. The recurrence rate is too high and dyspareunia or complete failure of coital function thereafter is much too common (Loxton, 1955, 1961).

It is suggested that the operation of posterior colpoperineorrhaphy should be performed more in accordance with the anatomical facts as described by the anatomists. I have checked these facts on innumerable occasions on physical examination of the healthy young nulliparous patient, at operation and by dissection of suitable subjects in the autopsy room. The formalinized, hardened and injected cadaver of the anatomical dissecting room, although forming the basis of all anatomical study, is by reason of its extreme age, atrophy and interstitial fibrosis, a much less suitable subject for detailed study of this region.

It has been said that the term posterior colpo-perineorrhaphy is cumbersome and should be abandoned in favour of such terms as posterior repair, repair of the pelvic floor or union of the levatores ani; but I know of no other term which so accurately and so completely describes what should be done at this operation.

The Surgical Anatomy.

The perineal body is that portion of the perineum or pelvic floor which is bounded by the vagina in front, the anal canal and rectum behind, the ischio-rectal fossæ on either side, and the skin of the perineum below (see Figure II). It is roughly pyramidal in shape, measuring about two inches in height, one and a half inches at the base from before backwards, and about the same from side to side. It consists mainly of muscle and fibrous tissue. The vertex and posterior two-thirds are composed mainly of muscle of both the striped and plain variety, whereas the anterior third, excluding the vertex, is composed chiefly of fibrous tissue.

The vertex, which is continuous with the recto-vaginal septum, consists mainly of the junction of the two partes pubo-rectales of the pubo-coccygeal portions of the levatores ani. This is the only point in the perineal body where the levatores ani as such are in apposition. They can be

FIGURE II.

readily seen approaching one another as a band of parallel fibres about half an inch in width, extending posteriorly, inferiorly and medially to constitute the ano-rectal ring.

Directly inferior to the junction of the partes pubo-rectales is a broad expansion of interlacing muscle fibres continuous with it, but running mainly transversely to form the deep external sphincter ani muscle. This expansion occupies about one-third of the perineal body. Inferior to this again is the superficial external sphincter ani, the perineal membrane on which lie the superficial transverse perinei and bulbo-spongiosus muscles covered by their fascia, the membranous and fatty layers of the superficial fascia and the skin.

The posterior wall of the perineal body consists of the longitudinal and circular muscle coats of the anal canal and rectum, including the internal sphincter muscle, covered by their submucosa and mucous membrane and, below the white line, the skin. The anterior wall is bounded by the endopelvic connective tissue covered by the stratified squamous epithelium of the vagina.

The Objects of this Operation.

To cure without deformity or loss of function must ever be the object of surgery. This ideal is not always attainable, but in the surgical treatment of prolapse it approaches fulfilment more closely than in the case of other surgical procedures. To the skilled surgeon who really knows and understands anatomy, and when the risk of sepsis can be excluded, the repair and permanent cure of genital prolapse are not difficult. The real problem is to perform the repair without producing deformity or incurring loss of function. It is here that the main difficulty lies.

The vagina has two principal functions—namely, coitus and child-bearing. Both these should be preserved whenever possible. The latter function (although, of course,

the more important) is limited by such factors as age, or made undesirable by economic circumstances. The former function, speaking in general terms, is subjected to far less limitation by reason of age or other conditions.

As the endopelvic connective tissue supports are the key to repair, so the preservation of all vaginal skin is the key to the retention of full vaginal capacity and therefore coital function. As a supporting structure in prolapse the vaginal skin plays no part and for this purpose it should be disregarded. As the most important means of ensuring adequate vaginal capacity after the repair of prolapse it should be retained in principle in its entirety (Loxton, 1955, 1961).

The Operation.

The incision is made in the mid-line in the posterior vaginal and perineal skin (Figure III). It commences at a point about one inch above the fourchette and ends about half an inch anterior to the anal margin. It is carried down through the skin and underlying connective tissue to the fibro-muscular tissue of the perineal body. The perineal and vaginal skin, with the underlying connective tissue attached, is then dissected laterally on either side and separated from the remainder of the perineal body for an extent which will allow complete anatomical reconstruction of this structure. In the process the attachments of the perineal membrane and levatores ani to the vagina are divided. Any scar tissue which can be identified as such is removed.

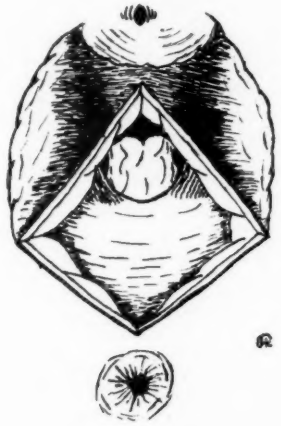

FIGURE III.

FIGURE IV.

The dissection of the posterior vaginal wall is continued upwards and it is separated in its whole thickness from the deeper structures, the surgeon cutting with the scalpel or scissors against the fingers of his other hand placed within the vagina. In this way it is easier to gauge the thickness of the vaginal wall and avoid injury to the anal canal and rectum. At a variable distance above the fourchette a point will be reached at which the connective tissue beneath the posterior vaginal skin, or in other words the recto-vaginal septum, is attached to the longitudinal muscle fibres of the outermost coat of the rectum. This is the recto-vaginalis muscle, homologous with the superior and inferior recto-urethralis muscle in the male. It must be divided by sharp dissection; when divided, access will be afforded to a space between the rectum behind and the recto-vaginal septum in front lined by a shining membrane, the fascia of Denonvillier (Figure II). This space extends to the limits of the pouch of Douglas and is opened as far as is necessary to deal with the defect in the recto-vaginal septum.

With downward traction on the anal sphincter, the upper surface of the levatores ani clothed with their deep fascia is brought prominently into view (Figure III). By a sweep of the finger or the finger covered by a gauze sponge the rectum is separated on either side from the upper surface of the levatores ani and its fascia, as far laterally as will allow accurate reconstruction of the apex of the perineal body and restoration of the normal ano-rectal ring.

A series of three simple interrupted sutures of number two plain catgut are placed in the musculo-fascial tissues of the perineal body as they lie on either side of the rectum and anal canal; the surgeon proceeds from above downwards, picking them up as close as possible to, but without entering, the latter (Figure IV). A series of two sutures are placed similarly, but in a plane anterior to the former (Figure V), and finally a third is placed more anteriorly still. The sutures are not tied at this stage, since tying would restrict access, but are left long and secured by hæmostatic forceps. When these three layers of sutures are eventually tied, the effect will be to reconstruct the perineal body in its normal pyramidal shape.

Attention is now directed to the repair of any defect in the recto-vaginal septum resulting in rectocele. It is not necessary, nor is it desirable, to extend the mid-line incision in the vaginal skin in order to do this. The septum can be repaired with the skin attached. A series of simple interrupted sutures of number 00 chromic catgut are placed from above downwards in the posterior vaginal wall. The sutures are placed by means of a curved, non-cutting needle, which enters the vaginal skin a few millimetres lateral to the mid-line (Figure VI) and then passes laterally for an appropriate distance (often several centimetres) through the substance of the recto-vaginal septum before emerging from it. The needle is then carried round and made to enter the recto-vaginal septum at a similar point on the opposite side. After again passing through the substance of the recto-vaginal septum it is made to emerge through the vaginal skin close to, but a few millimetres lateral to, the mid-line, on the opposite side to its point of entry. It is convenient to tie these sutures from above downwards as soon as they are placed. The distribution of these sutures depends on the shape and extent of the septal defect. As the lowermost sutures are tied the mid-line wound in the vaginal skin is closed (Figure VII). In the tying of the more widely placed sutures the stretched and attenuated endopelvic connective tissue of the recto-vaginal septum is drawn together into a solid, supporting structure (Figure VI). The attached vaginal skin becomes furrowed, wrinkled and apparently redundant. For reasons mentioned elsewhere (Loxton, 1955, 1961) it is not removed.

The three layers of interrupted sutures previously placed in the perineal body and secured with forceps are now tied from above downwards and from behind forwards. The pyramid-shaped space between the rectum and vagina created by the previous manœuvre is now filled by the solidly united fibro-muscular tissue of the perineal body.

The perineal membrane and the superficial perineal muscles with their fascia, which have been exposed by separation of the vaginal perineal skin and connective tissue flaps, are now united by a series of interrupted

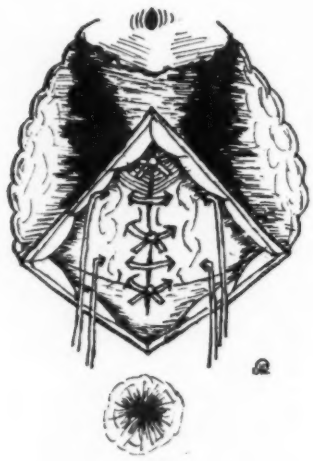

FIGURE V.

number 0 plain catgut sutures commencing at the new fourchette and continuing posteriorly parallel but deep to the perineal skin (Figure VII). As these sutures are tied and the divided, stretched and attenuated superficial structures are drawn together medially from their former lateral positions, the previous concave or scaphoid appear-

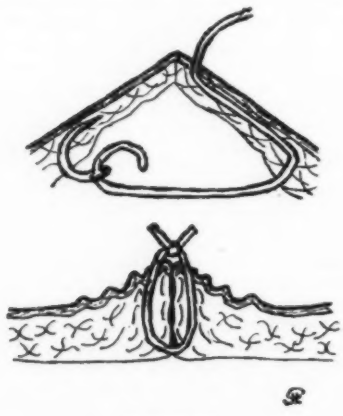

FIGURE VI.

ance of the perineal body disappears and is replaced by the normal flattened or slightly convex appearance. The perineal skin is closed from before backwards (Figure VIII) by a series of interrupted number 000 chromic catgut sutures so placed as to pick up the deeper tissues.

If at this stage two fingers of one hand are placed in the vagina and one finger of the other in the anal canal with palmar surfaces opposed, the thickness and triangular shape of the reconstructed perineal body can be readily appreciated. Provided that the uppermost plain catgut suture has been accurately placed in the partes pubo-rectales of the pubo-coccygeal portions of the levatores ani, at the apex of the perineal body (and this is the only suture which should unite the levatores ani proper), then the normal ano-rectal junction, which is palpable as a ring, will be found to have been reestablished. This is an important point in the correct anatomical reconstruction of the perineal body.

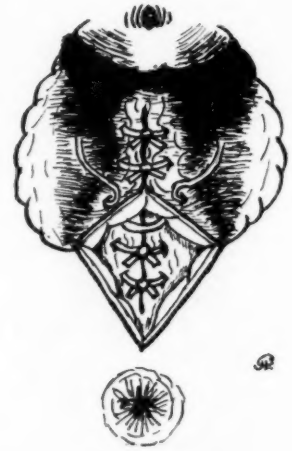

FIGURE VII.

In order to obliterate dead spaces, to avoid the accumulation of blood and serum and to press down firmly the vaginal skin and fascia onto the deeper structures, the surgeon then packs the vagina firmly with several yards of three-inch gauze. Because of this it is necessary to

FIGURE VIII.

drain the bladder per urethra by means of a catheter so long as the gauze is in place. Both of these are removed after 24 hours. Finally a finger is placed in the anal canal and rectum; this is important and should never be neglected. The anatomical reconstruction is checked. A deliberate search is made for any of the deeper sutures which may have been placed inadvertently through the anal or rectal mucous membrane. In a detailed anatomical reconstruction such as the one described, the likelihood

of this occurring is greater than in the older methods, though even in these the risk of damage to the bowel is still appreciable. Should a suture be found to have entered the lumen of the bowel, it can be dealt with simply; it is divided with scissors and its cut ends are allowed to retract from the mucous membrane into the deep tissues, where they can do no harm. This accident has occurred in a number of my earlier cases, but no harm has ever resulted, provided that it has been recognized and dealt with as described. An antibiotic cover has usually been given for the first three post-operative days, although I doubt very much now whether this was necessary.

The patient is allowed to get out of bed usually about 24 hours after the operation, when the gauze and catheter have been removed; provided that the repair has been performed adequately, it will stand up to the ordinary stresses and strains of post-operative early ambulation. She is allowed to go home as soon as the wound has healed—this is usually by the tenth or twelfth post-operative day. Intercourse is avoided for six weeks after the operation, by which time healing is usually complete.

Results.

So far 421 posterior colpo-perineorrhaphies have been performed by myself more or less in the manner described. The base line was taken some years ago, when it was realized that the operation which had gradually been evolved at that time differed so much both in principle and in technical detail from the standard procedures as to warrant its inclusion under the heading of an entirely new operation.

The results have been eminently satisfactory. So good have they been that, although this technique is more difficult than the standard procedures and somewhat more time-consuming, I have seen no reason to vary it except in the direction of attention to even greater detail.

The carrying out of a strict and accurate follow-up of so large a series of cases would be a task of considerable magnitude to a surgeon in private practice without additional finance or a department experienced in research at his disposal. Should such a survey be undertaken, then it should be conducted by one surgeon, who should personally examine all patients, have access to all records, be capable of arriving at an unbiased opinion and classify all results in accordance with a recognized formula. However, I have no knowledge that any such formula at present exists. I would suggest therefore a classification along the following lines, which would be applicable to all types of prolapse: (i) prolapse of the first instance (original prolapse); (ii) prolapse of the second instance (recurrent prolapse): (*a*) recurrence of the original prolapse in spite of operation, (*b*) development of fresh prolapse, in part or parts of the genital tract other than that previously repaired. With regard to its extent the prolapse should be designated as first, second or third degree, depending on whether (as in the case of hæmorrhoids) under the influence of gravity or raised intraabdominal pressure, it extends down to or through or remains persistently outside the particular aperture or introitus involved.

In the absence of such a survey I can state that all patients have been followed up from a period of three months to more than five years. There have been no recurrences of the original prolapse of such an extent as to warrant further operation. There has been one case of fresh prolapse—namely, true rectocele—which was either not recognized at the time or occurred *de novo* in a part of the posterior vaginal wall not previously repaired. In about 10% of cases there has been some sloughing of the subcutaneous perineal tissues apparent at about the seventh to tenth day, resulting in opening up of the perineal wound and, after separation of the slough, healing by second intention over the next seven to ten days. Although this has resulted in some deficiency in the subcutaneous tissue, it has not affected the other fibro-muscular components of the perineal body.

Prolapse, unlike cancer, does not require a thorough detailed and prolonged follow-up. If a prolapse is going to recur, evidence of this is frequently apparent, if it is really looked for, by the time the patient leaves hospital, or at the most some three to six months thereafter.

Conclusion.

It is the duty of a surgeon to exercise at all times a critical faculty in the assessment of his own work and the work of others. Without the exercise of this faculty in an objective and unbiased manner, surgical results are unlikely to improve. Even the simplest and most commonly performed surgical procedures unquestionably benefit from such examination.

Summary.

Posterior colpo-perineorrhaphy, an operation performed for more than 50 years without significant improvement, can no longer achieve the standard of results demanded by modern surgery.

The reason appears to be the wide discrepancy between the concept of the anatomy of the region described by the anatomist and that held by the surgeon.

The perineal body is a pyramid-shaped structure in relation to the lower third of the posterior vaginal wall. The levatores ani as such play little part in its composition, being in apposition normally only at its apex.

An operation is described in which emphasis is placed on accurate reconstruction of the perineal body. The levatores ani are united at one point only—namely, at its apex—with reconstitution of the normal ano-rectal ring.

The repair of prolapse in accordance with anatomical principles, and at the same time the importance of the preservation of full vaginal function, is emphasized.

The results of 421 such operations performed by the author are briefly discussed and a formula for a more detailed follow-up is outlined.

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Reports of Cases.

REPORT OF A CASE OF MYXOMA OF THE LEFT ATRIUM.

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THE aim of this report is to present a case of a myxoma of the left atrium and to reemphasize certain features that may be of value in the successful early diagnosis of such a case, in the hope that, in this day of safe cardiac surgery, more of these unfortunate people may be diagnosed and adequately treated before death. A review of the subject precedes the case report. This review has been deliberately made brief, for excellent articles on this topic have already been written by Mahaim (1945), Pritchard (1951), Bahnson and Newman (1953) and others. Cooley, Morris and Attar in 1959 published a report on a series of cases treated surgically.

Review.

Myxomata of the heart, although rare, comprise about 50% of primary cardiac tumours. Almost all have been found within the atria, 75% occurring in the left atrium. Other sites of this tumour have been reported—for example, in the left ventricle or attached to the valve of the inferior vena cava—but such cases appear to be much less common. Myxomata occur in persons of all ages, including infants during their first year of life,

but the majority of patients are between 30 and 60 years of age (Pritchard, 1951). Pritchard, in his series of over 100 cases, found the sex incidence to be equal, but others have found this tumour to be commoner in females.

An interesting point in the ætiology of these myxomata is that the great majority are attached to the fossa ovalis. The tumour usually fills its respective atrium and gives rise to symptoms by producing some degree of obstruction to the mitral or tricuspid valves. In his review on cardiac tumours Pritchard gives an excellent description of the pathology of these neoplasms. The following is a summary of his findings. They are usually polypoid and often pedunculated, their external surface being smooth or gently lobular. They may be white, yellow or light brown in colour, and often display areas of hæmorrhage. Thrombi sometimes overlie the surface of the tumour. Microscopically the average tumour is poorly cellular, covered by unbroken endocardium and composed of loose tissue of varying vascularity. Lymphocytes, plasmocytes, sporadic stellate cells and giant multinucleate cells are scattered throughout, but mitoses are not usually seen. Blood vessels are usually of capillary size, but larger vessels with thick walls are frequent and occasionally thrombosed. Hæmorrhages into the stroma are common, and hæmosiderin is found in varying amounts, both free and in macrophages. Variable amounts of mucin, elastic tissue and collagen comprise the matrix. Bone and marrow tissue and cysts lined by endothelium have been described (Pritchard, 1951).

The origin of intracardiac myxomata is still uncertain. Although a number of investigators have felt that these tumours are only simple intracardiac thromboses, the general opinion these days is that these tumours are true benign neoplasms, probably arising from the subendocardial connective tissue, especially in the region of the fossa ovalis.

Clinically, myxomata of the left atrium have closely resembled mitral stenosis in their manifestations. Myxomata of the right atrium may simulate tricuspid stenosis, constrictive pericarditis or the superior vena caval syndrome (Cooley *et alii*, 1959). Embolization with a heart murmur and a low-grade fever may simulate subacute bacterial endocarditis. Arrhythmias such as auricular tachycardia or fibrillation may also occur in association with myxomata. Possible distinguishing clinical features may include: (i) heart murmurs varying with changes in posture; (ii) sudden syncope and possible epileptiform attacks due to cerebral anoxia following sudden occlusion of the mitral orifice by the tumour, in a patient suspected of having mitral valve disease, especially if fibrillation or other arrhythmias are absent at the time; (iii) heart failure which is sudden and refractory to digitalis and diuretics in a patient with suspected mitral or tricuspid valve disease; (iv) the preference by the patient, although in heart failure, for a position in which the head is lower than the thorax; (v) the occurrence of peripheral embolic phenomena, unaccompanied by auricular fibrillation, in a patient suspected of having mitral valve disease; (vi) the occurrence of emboli, but without splenomegaly or positive blood cultures, in a patient with a heart murmur and low-grade fever suspected of having subacute bacterial endocarditis; (vii) the occurrence of pulmonary emboli in a suspected case of tricuspid disease, but without evidence of auricular fibrillation or venous thrombosis of the limbs (although, of course, venous thrombosis of the limbs may occur without clinical symptoms or signs).

Up to 1951 no myxoma had been diagnosed during the life of the patient. Although most of these tumours recognized during life in the past 10 years have been discovered at mitral exploration for mitral stenosis, diagnosis may also be established by angiocardiography and cardiac catheterization. Cooley *et alii*, in their article, mention the findings in these special investigations that may make the diagnosis of the tumours possible before cardiac surgery (Cooley, Morris and Attar, 1959).

Excision of the tumour by the cardio-pulmonary by-pass technique appears at present to be the most valuable and successful method of treatment of these neoplasms.

Cardiac myxomata are discovered more rarely still in children. Because the following case report deals with a child, brief mention shall be made of two examples of this tumour occurring in children.

The first case is that of a boy, aged three years and three months, reported by Centeno in 1955. This child's illness simulated rheumatic heart disease, and he died three months after admission to hospital, the diagnosis of myxofibroma of the left atrium being made at autopsy. The outstanding clinical feature in this boy's illness was that, although in heart failure, he preferred the prone position with his head lower than his trunk (Centeno, 1955).

The second case to be mentioned is one reported by Chao, Henry and Rosenberg in 1960. The patient was a girl, aged nine years, who presented at first with acute left internal carotid artery occlusion, proved by carotid arteriography. While she was in hospital an apical diastolic murmur, which varied from day to day, was noted. Later during the course of the illness she developed congestive cardiac failure, which proved resistant to treatment. Death occurred suddenly after 31 days, but during the week prior to death, clumps of unusual and unidentifiable cells in blood smears were noted and the possibility of an intracardiac tumour was suggested. Autopsy showed the presence of a left atrial myxoma, a large thrombus in the left internal cartoid artery and focal areas of infarction in both kidneys and spleen (Chao, Henry and Rosenberg, 1960).

Several other reports of cardiac myxomata in children may be found in the literature.

Clinical Record.

On February 10, 1960, a boy, aged six years and ten months, was admitted to the Royal Alexandra Hospital for Children with a six weeks' history of lassitude, pallor, loss of weight and anorexia. He had been previously energetic and full of life, apart from an "asthmatic" attack nine months prior to his admission. On examination, he was found to be a pale, ill child and to have a regular tachycardia of 120 beats per minute, a diffuse and thrusting apex heat, apical pansystolic and diastolic murmurs, engorged neck veins, a large, tender liver and an axillary temperature of 97° F.

Laboratory investigations revealed a blood sedimentation rate of 6 mm. in one hour by the Westergren method, a hæmoglobin content in the blood of 14·2 grammes per 100 ml. and a white-cell count of 18,000 cells per cubic millimetre, of which 74% were neutrophils; from a swabbing of his throat was cultured a group A hæmolytic streptococcus. An electrocardiogram showed the presence of *P* mitrale, *P* pulmonale, a prolonged *P-R* interval and some evidence of hypertrophy of both ventricles. A provisional diagnosis of acute rheumatic carditis was made, and the patient was digitalized and treated with corticosteroid therapy, a salt-free diet and sedation.

His subsequent history may be divided into (i) the initial stage, (ii) the stage of embolization, (iii) the stage of resistant cardiac failure, (iv) the stage of deterioration and (v) autopsy findings.

Initial Stage: February 11 to May 16.

Within two days of his admission to hospital the patient's condition improved a great deal. His pulse rate slowed to 48 to 60 beats per minute and remained regular. The jugular venous pressure was no longer raised and the liver was no longer palpable. A loud first heart sound and a short mid-diastolic murmur were noted over the mitral area and the blood pressure was 100/70 mm. of mercury. A second electrocardiogram showed sinus bradycardia (believed to be a response to steroid therapy), but otherwise the tracing was normal and accordingly the dose of corticosteroid was reduced.

The impression at this stage was that signs of established disease of the mitral valve were present, but that the general cardiac status was satisfactory.

The patient remained well until May 17, except for (i) an episode of severe precordial pain on March 4 associated with a transient pericardial friction rub, and (ii) recurrence of cardiac failure on March 25, at which time the liver once again became palpable and pulsatile, the jugular venous pressure became raised and loud first and third heart sounds appeared in the mitral area, accompanied by the disappearance of the apical systolic murmur and a diminution in intensity of the apical mid-diastolic murmur. The patient was redigitalized and again placed on a low-salt diet. Within the next few days his condition once again improved, allowing reduction in the digitalis dosage.

Radiological examinations of the chest during this intial stage showed moderate cardiomegaly with enlargement of the left auricle. Progress electrocardiograms showed a reappearance of *P* mitrale and some evidence of hypertrophy of both ventricles.

Throughout this time the patient remained afebrile, but was given penicillin orally as a prophylactic measure.

Examination during the early part of May revealed a reappearance of the pansystolic apical heart murmur, the presence of a third heart sound in the mitral area and the absence of the apical diastolic murmur. The hæmoglobin content of the blood was 15·0 grammes per 100 ml., the white-cell count was 19,000 cells per cubic millimetre, of which neutrophils comprised 84%, and the blood sedimentation rate was 12 mm. in one hour by the Westergren method. His corticosteroid and digitalis dosages were reduced. The diagnosis of fibroelastosis was considered, but thought unlikely because of normal *T* waves on the electrocardiogram.

Stage of Embolization: May 17 to July 18.

On May 17 the patient suddenly became irritable, restless and semidelirious. Examination showed absent pulses in, and coldness of, the left upper and both lower limbs and also loss of speech. However, his pulse was regular and its rate was not increased. There was a loud first heart sound in the mitral area and a very loud second heart sound in the pulmonary area as well as a pansystolic apical murmur. The blood pressure remained 100/70 mm. of mercury, but the electrocardiogram showed considerable right ventricular strain and a persistence of *P* mitrale. The electroencephalogram showed diffuse changes in the brain, but with no localization.

The diagnosis was made of multiple emboli associated with rheumatic mitral valve disease and involving the aortic bifurcation and the cerebral and left radial arteries. Heparin and phenindione anticoagulant therapy was commenced, and administration of corticosteroids, digitalis and penicillin was continued. During the next five days the pulses in each of the affected limbs became palpable, but were diminished in the right lower limb; also a right facial paresis and a right hemiparesis became apparent, the latter being more marked in the lower limb.

Later still, the cardiomegaly became more marked clinically, with both right and left ventricular and pulmonary artery pulsations, very loud mitral first and pulmonary second heart sounds and pansystolic and mid-diastolic apical murmurs. A low-grade fever appeared after the embolic episode and bacterial endocarditis was considered, but repeated blood cultures produced no growth. The hæmoglobin content of his blood at this stage was 11·5 grammes per 100 ml., the white-cell count was 11,000 cells per cubic millimetre, of which neutrophils comprised 75%, and the blood sedimentation rate had risen to 103 mm. in one hour by the Westergren method.

The diagnosis at this stage was rheumatic heart disease affecting the mitral valve and producing predominantly incompetence, but also some stenosis, of this valve. Continued long-term anticoagulant therapy was considered preferable to left auricular appendicectomy at this stage because of the probable necessity of other cardiac surgery in the near future. Arrangements were made for cardiac catheterization.

It is fair to say that the diagnosis of myxoma of the left atrium was suggested, but it was rejected in favour of rheumatic heart disease.

Stage of Resistant Cardiac Failure: July 18 to July 27.

On July 18 the signs of heart failure reappeared—namely, a tender, pulsating liver, a raised jugular venous pressure, increased lethargy and a loose cough.

During this period the low-salt diet was continued, the dose of digitalis was increased, and chlorothiazide administration with potassium chloride was commenced—all without improvement.

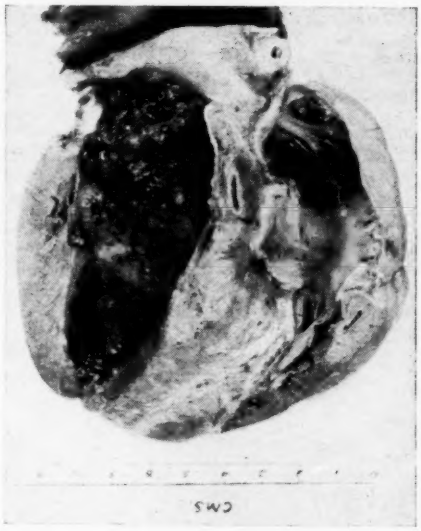

FIGURE 1: Posterior aspect of the heart. The left auricle is filled by tumour tissue which can be seen to project in the form of a cone into the left ventricle.

At this stage the apical diastolic murmur was no longer apparent. Examination of his blood for lupus erythematosus cells gave negative results, and the blood sedimentation rate of 3 mm. in one hour by the Westergren method had returned to within the normal range.

Opinion at this time was in favour of rheumatic heart disease with mitral incompetence but minimal stenosis and further complicated by pulmonary hypertension, tricuspid incompetence and right heart failure.

Stage of Deterioration Leading to Death: July 28 to August 5.

From July 28 to August 5 his condition deteriorated rapidly and was marked by dyspnœa at rest, oliguria, heavy proteinuria and a further increase in the jugular venous pressure and in the size of the liver. His blood pressure was 105/60 mm. of mercury, and the chest appeared clear clinically. A further electrocardiogram showed abnormally broad and tall *P* waves, right axis deviation and right ventricular preponderance. On July 30 the patient became puffy about the eyes and gained weight suddenly. The nephrotic syndrome associated with renal vein thrombosis was considered a possibility. The serum protein content was 6·3 grammes per 100 ml., of which albumin accounted for 3·2 grammes per 100 ml., but the blood urea content was 28 mg. per 100 ml. and the serum cholesterol content 140 mg. per 100 ml.

On August 4 he coughed up blood-stained sputum, and signs of fluid were obvious clinically over the right lower part of the chest. Œdema of the legs appeared and radiological examination of the chest showed opacities at both lung bases, as well as cardiomegaly. The patient's breathlessness increased and he died on August 5, 1960, six months after his admission into hospital and seven and a half months after the onset of his symptoms.

Autopsy Findings.

The right ventricle was hypertrophied and there was enlargement of all cavities of the heart, especially the right atrium. All the heart valves were entirely normal and their orifices were not dilated. Soft, red and amber, jelly-like tumour tissue was present in the left atrium, filling it and projecting through the mitral orifice into the left ventricle, occupying one-third of its cavity (Figure I). The tumour was found to have arisen from the left side of the interatrial septum over the fossa ovalis, this origin occupying an area 2 cm. in diameter. Microscopic examination of the tumour tissue showed it to be sparsely cellular, but containing much mucoid material, scattered, thin-walled vessels from which much hæmorrhage had occurred and clefts lined by surface endothelial cells, which had been invaginated during the growth of grape-like masses of tumour tissue (Figure II).

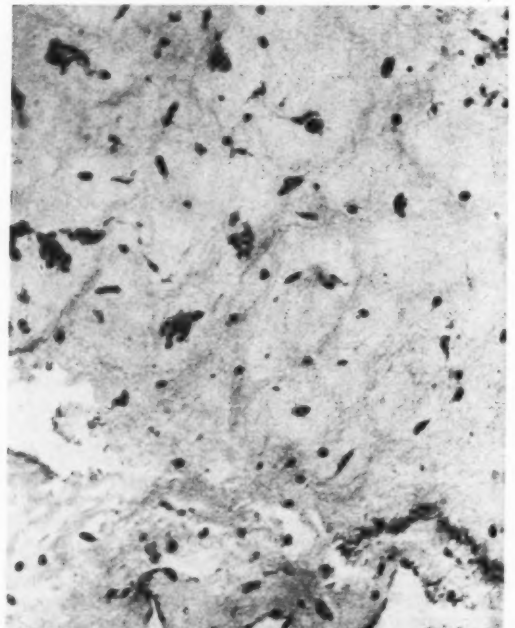

FIGURE II: Section of portion of tumour tissue showing the finely fibrillar and sparsely cellular structure. Elongated, flattened cells form a complete covering over the free surface (Masson's trichrome stain, × 400).

On examination of the lungs enlarged, tortuous vessels were noticed coursing for short distances in the pleura. Half of the right lower lobe, the left lower lobe and half of the lingula were dark red in colour and solid in consistency, with a dry cut surface, giving the appearance of massive recent hæmorrhages. On microscopic examination these hæmorrhages were found to be due, not to vascular thrombosis, but to necrosis of the small muscular arteries. This arterial necrosis with hæmorrhage was probably due to a sudden hypertension in the pulmonary circuit.

In the brain there was occlusion of the subsidiary branches of the middle cerebral artery on each side. Infarction of both cerebral hemispheres was present, involving the lower part of the parietal and the upper part of the temporal lobes, but the infarction was more marked on the left side.

Both liver and kidneys demonstrated chronic venous congestion, but the latter contained a number of infarcts, the cause of which was found to be tumour-tissue emboli. The spleen showed one infarct, also due to a tumour-tissue embolus.

Embolic, soft, amber, gelatinous material was found attached to the walls of both common iliac arteries.

The final diagnosis was made as follows: (i) myxoma of the left atrium; (ii) multiple infarctions of about the same duration, due to emboli arising from the intracardiac myxoma; (iii) large emboli found at the origins of the common iliac arteries and arising from the atrial tumour; (iv) arterial necrosis of the pulmonary vessels with massive hæmorrhages, probably due to a sudden hypertension in the pulmonary circuit.

Discussion.

This boy's history contains some of the features mentioned previously, namely: (i) a clinical picture of mitral valve disease; (ii) variable heart murmurs; (iii) embolization in the absence of auricular fibrillation; (iv) sudden, unexpected bouts of heart failure, in the final episode of which he failed to respond to therapy; (v) some clinical features of bacterial endocarditis, but without splenomegaly or positive blood cultures; (vi) a normal blood sedimentation rate during his illness except during the stage of embolization.

It is possible that the "asthmatic" attack nine months prior to admission was an episode of acute paroxysmal dyspnœa produced by temporary obstruction of the mitral valve by the tumour.

Perhaps cardiac catheterization with concurrent angiocardiography, performed while the patient was reasonably well during the early part of his illness, in order to assess the exact nature of the mitral lesion, might have provided an early diagnosis in time for treatment. Also, left auricular appendicectomy performed as soon as possible after the embolic episode, especially in view of the absence of auricular fibrillation, might have given the diagnosis. However, it is easy to be wise after the event.

It is hoped that this case report may be of some benefit in assisting in the early diagnosis and treatment of such a case, even if it only stimulates an awareness that such a lesion may be present. It may be wise to perform early cardiac catheterization and angiocardiography in any patient suspected of having mitral or tricuspid valve disease, if the clinical picture is in any way atypical.

Summary.

A case of myxoma of the left atrium occurring in a male, aged six years, has been reported with special reference to its pathological features and to the clinical features by which it presented. The report is preceded by a brief review of the literature on this subject. Finally, a summary is made of certain clinical features occurring in this case that may be of value in the diagnosis of the condition during life. It is suggested that cardiac catheterization and angiocardiography should be performed early in the case of any patient suspected of having mitral or tricuspid valve disease if the clinical picture is in any way atypical.

Acknowledgements.

I wish to thank Dr. S. E. J. Robertson for his permission to publish this case report and for his valuable help and advice during its preparation. I also wish to thank Dr. D. Stuckey and Dr. R. D. K. Reye for reading through the report and offering helpful criticism. Dr. Reye, who performed the autopsy, very kindly made available the

photographs which appear with the report. The photographs were printed by Mr. C. Noble, for whose services I am grateful.

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TORSION OF THE SPERMATIC CORD IN THE NEONATAL PERIOD.

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TORSION of the testicle was originally described by Delasiauve in 1840; this description was ultimately followed in 1897 by the first report of torsion of the spermatic cord in a new-born infant. Whereas torsion of the testicle has become a well-documented clinical entity, occurring frequently in young adults and not uncommonly in childhood, torsion in the neonatal period is rare. In addition to its rarity, two aspects of this latter condition are of interest. Firstly, the torsion is supravaginal or extravaginal, so that the tunica itself is involved, in contrast to the more usual situation where the torsion is intravaginal. Secondly, in torsion of the testicle outside infancy, a predisposing anatomical abnormality is usually present; Johnson (1960) recently drew attention to this, commented on the frequency of a bilateral abnormality of the mesorchium, and made a "plea for bilateral exploration". This situation did not exist in the reported cases of torsion of the spermatic cord in infancy; in these the mesorchium always appeared to be normal on the affected side. Therefore, there is no reason to assume that, when torsion occurs in the neonatal period, there is an abnormality of the attachment of the testicle on the contralateral side.

The terminology adopted in describing torsion provides considerable confusion, and statements such as the following, made by Charnock and Riddell (1955), do not help: "Torsion of the spermatic cord . . . often goes under the misnomer of torsion of the testicle." For all practical purposes it would seem reasonable to describe the cases in the neonatal period under the heading "torsion of the spermatic cord", and describe the more frequent occurrence in the older age group as examples of "torsion of the testicle".

Clinical Record.

On August 1, 1960, a male child was born after a pregnancy which was described as "normal apart from mild hydramnios", and an uneventful labour. The birth weight was 3·86 kg. At birth a swelling in the right side of the scrotum was observed. The right hemiscrotum was distended to approximately three times its normal size, and a visible bluish discoloration was present. On palpation, the swelling was found to be confined to the scrotum, without an associated inguinal element; it was firm, but not hard, and appeared to be slightly tender. The swelling was irreducible and there was no impulse with crying or straining; it was not transilluminable. Physical examination otherwise revealed no abnormality, and, in particular, a normal left testicle was palpable.

A provisional diagnosis of a neoplasm or hæmatocele was made, and arrangements were made to transfer the baby from the nursery to a pædiatric institution. Before operation a third differential diagnosis was added—namely, torsion of the testicle—and at operation this was shown to be the correct diagnosis. The region was explored under general anæsthesia (halothane-nitrous oxide); through a right inguinal incision the external inguinal ring was exposed and the spermatic cord was found in a state of 720° of torsion in a clockwise direction (Figure I). This torsion was quite clearly supravaginal so that the tunica, as well as its contents, was involved in the twist (Figure II). On delivery of the contents of the right side of the scrotum into the wound, the tunica was found to be macroscopically gangrenous and markedly thickened. Incision of the tunica revealed a straw-coloured effusion, and the testicle and epididymis both appeared to be gangrenous. Orchidectomy was

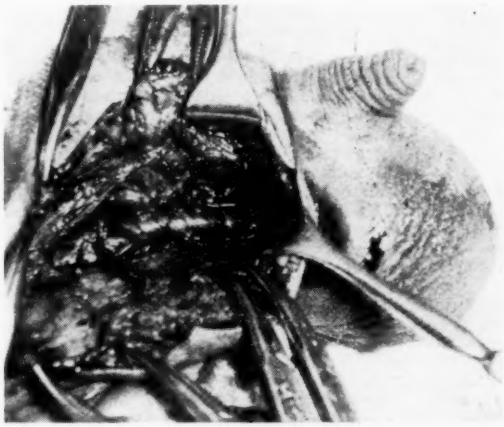

FIGURE I: Operative photograph showing torsion of the spermatic cord.

FIGURE II: Operative photograph illustrating supravaginal torsion with involvement of tunica in addition to contents.

performed by ligation and division of the spermatic cord at the level of the internal inguinal ring. An uneventful convalescence followed.

Discussion.

The case reported exemplifies the three main features of torsion of the spermatic cord in the neonatal period—namely, that the torsion is supravaginal, the condition occurs prior to birth and the ætiology is obscure.

Extravaginal Torsion.

With regard to the first of the three points—namely, the site of torsion—there is no doubt that extravaginal torsion is almost entirely confined to this particular age group. However, it has been stated dogmatically that this type of torsion does not occur; for example, Lowsley and Kirwin (1944) make the following comment: "The twist is invariably located in the free portion of the cord within the vaginal sac." Indeed it is difficult to explain the mechanism of supravaginal torsion; the most satisfactory explanation is based on the observations of Astley Cooper that the entire scrotal contents of the new-born infant can be lifted from the scrotum without tearing of tissue. There have been occasional reports of supravaginal torsion outside the neonatal period; one such report occurs in an article by Abeshouse. In his case, supravaginal torsion of an undescended testicle occurred in a child, aged 3 years. But Abeshouse agrees that "the extravaginal type is relatively rare", and states that "extravaginal torsion may involve the undescended or descended testes, but is more likely to be associated with the former". This statement would appear to have more significance in relation to torsion occurring outside the neonatal period.

Allen and Andrews (1940) reported a case in a baby, aged 10 months, and at the same time drew attention to the contention of Young and David that extravaginal torsion does occur. Actually, Young and David also pointed out that a condition analogous to supravaginal torsion is produced in animals by the procedure of bistournage, which is substituted in some parts of the world for castration. The bistourneur manipulates the testis so that it, with the epididymis and tunica vaginalis, is broken loose from the subcutaneous tissues, and then rotates it three or four times. An artificial torsion is thus created, followed by swelling and later atrophy.

Although extravaginal torsion has been reported outside the neonatal period, it is clearly very uncommon and furthermore does not produce a clinical and pathological entity as is found in the neonate.

Time of Torsion.

The similarity of the reported cases of torsion of the spermatic cord suggests that the torsion probably occurred in all of these at the same time. In the case reported in this article there is indisputable evidence that the torsion occurred prior to birth. Charnock and Riddell state that there is "pathologic evidence that the rotation took place in utero". It would seem probable that cases reported as occurring at the age of a few hours or a few days would, therefore, be examples of the common type, not noticed but present at birth. This could well apply to the first case reported in the literature, by Taylor in 1897. He referred to "the infant son of W.S." and stated:

> Nothing abnormal was noticed at the time of birth, but four hours later, the nurse called attention to the right testicle, which was found to be slightly larger than the left. By the next morning, the right testicle was twice the size of the left and appeared discoloured through the skin. It felt tense, round and smooth. There was no enlargement of the cord to be felt. The only thing possible seemed to be strangulation from some cause in or near the testicle. That night, the child was evidently in pain and cried incessantly while, towards morning, it had some slight convulsions. On the morning of September 19, Dr. Cave of Crewkerne saw the child with me and agreed to the necessity of castration. He gave chloroform and I removed the testicle and an inch of the cord. Testicle was round, smooth and very tense, the insertion of the cord appearing like that of the stalk of the apple. Mr. C. B. Lockwood, Assistant Surgeon to St. Bartholomew's Hospital, kindly examined the testicle for me and reported as follows:
>
> "The testicle and tunica vaginalis of a child. The spermatic cord is very thin and is twisted, apparently just above the tunica vaginalis. The whole cavity of the tunica and the whole testis and epididymis were full of coagulated blood."

Meredith Campbell reported two cases in 1948, commenting as follows: "My two cases are apparently the second and third to be reported in which the torsion occurred during the first day of life."

The premise that Taylor's and Campbell's cases were examples of intrauterine torsion is lent support by the largest series reported to date—namely that of Longino and Martin (1955). In their nine cases swelling was always present at birth, all the babies being born with "an enlarged firm reddish or purple non-tender mass which did not transilluminate".

Further support is obtained from other sources; thus Biorn and Davis reported two cases of torsion of the spermatic cord in the newborn. In one, the testicle was found "firmly embedded in adhesions" and they state that "the pathologist who examined the thickened capsule and the microscopic sections of the operative specimen estimated that the testicle had been infarcted for at least two weeks".

Peterson, too, has recently reported a case of extravaginal torsion of the spermatic cord. In his case, a pre-operative X-ray film had revealed calcification in the scrotal mass. This calcification was subsequently visible in the gross specimen removed at operation.

Ætiology.

No light has been shed on the ætiology of the condition under discussion. Intravaginal torsion is usually associated with a predisposing congenital anomaly; this takes the form of an abnormality of the mesorchium with consequent undue mobility of the testicle. Such abnormalities have not been reported in the cases of neonatal torsion. Whether the torsion precedes the onset of labour is, as yet, uncertain, and the part played by factors such as anoxia, trauma, cremasteric development and so on, is purely hypothetical.

In summary, therefore, supravaginal torsion of the testis is a recognizable clinical entity in the neonatal period, characterized by its pathological and clinical features and an unexplained ætiology. The available evidence suggests that the torsion precedes birth, and, although the condition is certainly uncommon, it may not be as rare as has been suggested by many authors—for example, Trillat (1940), who stated "the diagnosis made . . . was torsion of the testicle . . . despite the total absence of publications about this accident in the newborn".

Management.

Is Operation Necessary?

Although in the presence of the characteristic physical signs the diagnosis is virtually certain, it is difficult to justify a completely conservative régime, and the logical approach is to explore the mass. This is in disagreement with the approach suggested by Glaser and Wallis (1954), who point out the similarities between torsion and spontaneous hæmorrhagic infarction of the testicle in the newborn infant and report two cases in which no operation was performed.

What is the Correct Operative Procedure?

It is unlikely that gangrene resulting from torsion will be followed by suppuration, particularly if the torsion is reduced. Orchidectomy is therefore probably unnecessary, and apparently, despite the original appearance of gangrene, subsequent atrophy may be relatively minimal. It would therefore seem reasonable to untwist the cord and replace the testicle in the scrotum.

Is Fixation of the Other Testicle Necessary?

The evidence available so far suggests that the occurrence of torsion of the spermatic cord in the neonatal period does not in any way indicate any increased likelihood of torsion on the other side. The absence of anatomical abnormality on the affected side is in keeping with this evidence and operative fixation of the contralateral testicle is therefore probably unnecessary. Longino and Martin state that the other side was not explored in any of their nine cases, nor have there been any cases of torsion subsequently on the unexplored side. However, a case can certainly be made out for fixing the other testicle and no one could deny the wisdom of such a move, even if one did not oneself support such a view.

Summary.

1. A case of neonatal torsion of the spermatic cord is presented.

2. This condition, although rare, is a recognizable clinical and pathological entity with three main features.

3. The literature is reviewed and the management of the problem discussed.

Acknowledgements.

It is a pleasure to thank Dr. W. J. Rawlings, who referred the patient described, and to acknowledge the help of Miss R. Doig and Mrs. D. James, librarians at the Royal Children's Hospital, Melbourne, my secretary, Mrs. J. McAllister, and Mr. C. Murphy of the Department of Photography, Royal Children's Hospital, Melbourne.

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PEUTZ-JEGHERS SYNDROME: A CASE REPORT.

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THE syndrome of muco-cutaneous pigmentation and intestinal polyposis, now called Peutz-Jeghers syndrome, was first described by Sir Jonathan Hutchison in 1896. He described peculiar pigmentation about the lips and buccal mucosa in identical twin girls, one of whom subsequently died of intussusception at the age of 21 years. Peutz in 1921 first recognized the polyposis and pigmentation as a syndrome. However, it was due to the comprehensive review of Jeghers *et alii* in 1949 that the syndrome became well recognized as a clinical entity.

The case to be reported presents no new features, but emphasizes the need for examination of the skin in patients with abdominal pain and gastro-intestinal bleeding.

Clinical Record.

The patient, a male, aged 27 years, of French descent, arrived at the casualty department of the Royal Prince Alfred Hospital on January 8, 1961, complaining of severe, generalized abdominal pain of six hours' duration. For the past five years he had suffered from intermittent, colicky pain situated in his epigastrium and left hypochondrium. These attacks of pain lasted only a short while, were not severe and appeared to come on after meals. Antacids and milk gave him no relief, but analgesics were sometimes effective. On the day prior to his admission to hospital he felt unwell and vomited his evening meal. Approximately six hours later he developed severe, generalized abdominal pain. Following this he vomited small amounts of food on three occasions. The only stool he passed prior to admission was black, although formed.

Physical examination showed him to be a pale, olive-skinned, slightly built man with numerous small, light-brown to dark-brown spots on his lips, buccal mucosa, eyelids, forehead and fingers (Figure I). There was no

FIGURE I.

pigmentation elsewhere. Moderate finger clubbing was noted. His abdomen was slightly tender to palpation, but neither rigidity nor rebound tenderness was present. Melæna stools were found on rectal examination. When questioned specifically about his pigmentation, he stated that, as far as he could remember, the pigmented spots had been present all his life and that his mother also had a similar type and distribution of pigment. No further family studies could be carried out as he had no siblings and his parents were dead. Surgical correction of a scoliosis had been carried out some years previously. The hæmoglobin value was 8·2 grammes per 100 ml. and a barium-meal X-ray study revealed a dilated stomach with a large, mobile polyp in the second part of the duodenum (Figure II). A small-bowel barium X-ray series revealed no further polyps. At gastroscopy a number of small sessile polyps were seen in the distal third of the stomach. Sigmoidoscopy and barium-enema X-ray examinations gave normal results. He received a transfusion of two units of packed red cells. He complained of some colicky abdominal pain while in hospital and this continued up to the time of his discharge from hospital.

Discussion.

There are many excellent papers dealing with this syndrome in the literature (Peutz, 1921; Jeghers, 1949; Bartholomew, 1957; Dormandy, 1957), but a brief résumé of the condition may be of value.

[1] Supported by a grant from the Bushell Trust.

The symptoms are usually those of borborygmi or vague, colicky abdominal pain, which may be very severe, and many patients ultimately present with intussusception. The repeated attacks, experienced by the patient reported, are consistent with minor bouts of intussusception, and he further demonstrated the other major symptom of the condition—namely, gastro-intestinal bleeding.

The age of presentation varies widely, but most cases are diagnosed while the patients are in their mid-twenties. The disease may affect people of any race. There is no increased incidence, as originally thought, in people of dark complexion. Approximately 55% of patients have a positive family history, and this disease is apparently

FIGURE II.

transmitted as a Mendelian dominant with incomplete penetrance. The patient's suggestion that his mother had buccal pigmentation is interesting in this regard, but it is unfortunate that other members of the family are not available for study.

The common sites of pigmentation are the buccal mucosa, the orifices of the face, especially the mouth, and the extremities—for example, the hands, fingers, feet and toes (Peutz, 1921; Jeghers, 1949; Tanner, 1951; Savage, 1954; Bartholomew, 1957). It has also been reported on the rectal mucosa (Peutz, 1921), abdominal wall (Croue and Light, 1954) and hard palate and tongue (Bortz and Bethell, 1955). The pigment is melanin and has been observed at birth, but in most cases it is first noticed in childhood. Facial pigmentation is said to fade after the age of 25 years (Peutz, 1921). Freckles are unlikely to be confused, as they have an entirely different distribution and never occur on the buccal mucosa. In Addison's disease, pigment is not localized, but widely distributed, and is more pronounced in body folds and pressure areas.

The other major feature of the syndrome is polyposis. The polyps are invariably situated in the small bowel. Other common sites are the large bowel, rectum and stomach. Rarely polyps are described in the bladder, nose, appendix and even bronchus (Peutz, 1921; Bartholomew, 1957; Dormandy, 1957). The polyps are usually benign adenomas. They may be sessile or pedunculated and vary in size from microscopic up to 7 cm. in diameter. It was previously believed that the small-bowel polyps were prone to undergo a malignant change (Peutz, 1921; Jeghers, 1949), and it has been reported that this occurs in 15% to 20% of cases. Dormandy and Bartholomew subscribe to the view that the small-bowel tumours are hamartomas and are not premalignant (Dormandy, 1956, 1957, 1959; Bartholomew, 1957). Their hypothesis is based on the findings of smooth muscle, gastric secretory cells and Paneth cells within the tumours. This theory is also supported by the fact that no case has been reported of these tumours having given rise to metastases. This is in contrast to the behaviour of malignant small-bowel tumours in the absence of buccal pigmentation. Rintala (1959) adds further support to the hypothesis by describing the histological state of small-bowel polyps removed from a father and his two daughters in eight operations as typical of hamartomas. In the light of this evidence, Guillard's (1958) report that there is a relatively low DNA content and "weak nuclear activity" in the small-bowel polyps of the Peutz-Jeghers syndrome may be relevant.

However, the colonic polyps in the Peutz-Jeghers syndrome appear to be true adenomata, in that they are composed essentially of goblet cells. Dormandy in 1957 emphasized the need for careful observation of large-bowel polyps, as four of his 21 patients died from carcinoma arising in large-bowel or rectal polyps.

A further interesting feature of the Peutz-Jeghers syndrome is the occurrence of skeletal and soft-tissue abnormalities. Clubbing of fingers and toes and scoliosis are well documented, but it may be that the reported occurrences of osteomas and soft-tissue tumours—for example, sebaceous cysts—has arisen from a confusion of the Peutz-Jeghers syndrome with that described by Gardner (1952, 1953), in which multiple polyposis of the colon is associated with osteomas, epidermoid cysts and desmoids (Smith, 1958). It is unlikely that the Peutz-Jeghers syndrome will be confused with multiple familial polyposis of the colon, in which the polyps are predominantly colonic and buccal pigmentation does not occur.

The management of a patient with Peutz-Jeghers syndrome is conservative. However, Dormandy (1957) suggests local removal of small-bowel polyps which are causing symptoms. He believes that it is rarely necessary to perform emergency surgery, since the intussuscepted bowel rarely becomes strangulated.

Summary.

A case of intestinal polyposis with muco-cutaneous melanin pigmentation (Peutz-Jeghers syndrome) is described. The patient reported, a male, aged 27 years, of French descent, had experienced recurrent bouts of colicky abdominal pain for many years and was admitted to hospital with severe abdominal colic and melæna stools. He was found to have pigmentation around his mouth and on his eyelids, forehead, buccal mucosa and fingers, and stated that his mother had similar pigmentation. He had no siblings and his parents were dead. Gastric polyps were seen at gastroscopy and a large duodenal polyp was demonstrated on a barium-meal X-ray examination. No polyps were found elsewhere in the bowel. Moderate finger clubbing and scoliosis were present. The patient is discussed in relation to the recent literature on this subject.

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I would like to thank Dr. A. P. Skyring for assistance in the preparation of this paper; Mr. S. Woodward-Smith, of the Department of Medical Illustration, University of Sydney, for the photography; Dr. D. D. Stephen, of the Department of Radiology, for the radiological studies;

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SKIN ERUPTION IN A GROUP OF WATERSIDE WORKERS DUE TO A MITE, PYEMOTES VENTRICOSUS.

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A GROUP of 24 waterside workers was unloading a cargo of wooden boxes of tobacco from a ship at the Victoria Docks, Melbourne. The maximum daily temperature at the time varied between 75° and 93° F., and it was considerably hotter in the holds where the men were working.

After periods varying from 16 to 48 hours, 16 of the men developed a rash. Six of these men were examined. The eruption consisted of papules distributed on the trunk, buttocks and proximal parts of the limbs (Figure I). Some of the papules were surmounted by a small vesicle, and with some there was an associated urticarial element. No fever, proteinuria or other significant abnormality was found.

The clinical features were consistent with mite bites, and the material handled suggested that the mite was

FIGURE I: Eruption due to *P. ventricosus*.

FIGURE II: *P. ventricosus* (immature female).

likely to be *Pyemotes (Pediculoides) ventricosus*. This is a not uncommon ectoparasite of many types of insects, particularly those which attack stored products. Its

detection requires careful examination, as the mobile stages are only about 0·25 mm. long.

In samples of dust collected from one of the holds of the ship, many dead beetles were found and identified as the tobacco beetle *Lasioderma serricorne*. Living and dead larvæ of this beetle were present in the tobacco leaf. Microscopic examination showed *P. ventricosus* (Figure II) to be present in the dust, and one specimen was found in a leaf, feeding on a larva of the tobacco beetle. This beetle has often been recorded as a host of the parasite.

The holds of the ship were treated by the method of fogging, using gammexane and pyrethrum. Unloading was then continued, and no further mite attacks were reported.

Comment.

Straw, grain, peas, beans, cotton seed, tobacco, hay, grass and broom corn are among the substances which may harbour the mite. The skin eruption caused by the mite is variously known as "grain itch", "straw itch", "prairie itch", "hay itch", "barley itch", "cotton-seed itch", and "mattress itch". The condition is an occupational hazard of certain activities, and it has been reported in farmers, millers, bakers, dock workers, tobacco workers, packers, broom-factory workers and others. It often occurs in epidemic outbreaks. In Australia, operations such as chaff cutting or those which involve the handling of hay from stacks a year or more old have been followed, on occasion, by the skin eruption (Swan, 1934), and it has also been reported in persons sleeping on mattresses containing straw infested with *P. ventricosus*.

The incidence of bites appears to be related to the temperature of the environment.

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Reviews.

Protection Against Radiation: A Practical Handbook. By John D. Abbatt, M.B., Ch.B., D.M.R., J. R. A. Yakey, Ph.D., B.Sc., A.Inst.P., and D. J. Mathias; 1961. London, Melbourne, Sydney, Toronto, Cape Town, Auckland: Cassel & Company Ltd. 8¾" × 5½", pp. 200, with illustrations. Price: 25s. net (English).

THIS book is intended as a guide for industrial medical officers who are responsible for the health of workers engaged in radiation work. Personnel protection, working conditions, shielding against radiation and the medical care of radiation workers are, therefore, the principal subjects, and half the book of some 200 pages is given to the management of these problems. Dr. Abbatt is the Chief Medical Officer for the General Electric Company and has first-hand experience of these matters in its Atomic Energy Division. The remaining pages are restatements of the basic physics of radiation, radiation dosimetry and radiation detection, to allow intelligent discussion of radiation hazards and maximum permissible doses and levels of radiation. A feature of this section is the use made of nomograms. Their number and scope are remarkable. If simple rules are followed, handy first approximations can be made. In realms hitherto left to the physicist, this approach must be useful to the industrial physician. References follow these compressed guides to the necessary data for more detailed and absolute calculations. It is indeed a handy and well-presented practical work in the radiation-protection field of preventive medicine.

Clinics in Electrocardiography. By Dale Groom, A.B., M.D., M.S. (in Med.), F.A.C.P.; 1960. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 10" × 6¾", pp. 152, with illustrations. Price: 64s. (English).

THIS slender, well-produced volume, aimed at the senior medical student and the early post-graduate, originated from a series entitled "The Electro-Cardiogram of the Month" published in *The Journal of the South Carolina Medical Association*, and the author attempts to correlate a particular graph with its clinical counterpart. This can, at best, be only partly successful, because of the non-specific nature of many electrocardiographic findings, so that after dealing with the few electrocardiograms that are fairly pathognomonic—for example, basic disorders of rhythm and infarction—the author finds himself in shifting sands. Thus he merely illustrates some interesting case findings—for example, fibroelastosis, cardiac trauma, amyloid disease and neoplasm of the heart, tracings of no didactic value.

The best sections are those on the arrhythmias, and the author is bold enough to discuss functional *T*-wave changes and the effect of emotion on the electrocardiogram. This important subject is too controversial to allow conclusions about it to be drawn, and more work is needed in this field. The effects of electrolyte changes are presented, but not enough cases are illustrated to round out the picture. The chapter on "The Stress Electro-Cardiogram" is illustrated by a case which breaks two of Master's cardinal rules: the pre-exercise graph was abnormal, and there was a clinical suggestion of recent infarction.

Congenital heart disease, apart from dextrocardia and rudimentary right ventricle, is not discussed, despite the fact that a septum primum lesion gives an almost pathognomonic graph.

In the discussion on precordial *R'* waves in right chest leads no conclusions are reached. The author omits to mention than an *R* wave in V1 can be due to strictly posterior infarction, perhaps deliberately so, because the unipolar theory of an "electrical window" situated posteriorly cannot explain such an *R* wave in lead V1, whereas forward displacement of the *QRS* vector loop due to loss of posterior potentials can and does explain it.

This book can be read in an hour, and is useful for the basic disorders. It is a model of clarity in presentation, but its lack of completeness is a disadvantage.

Fearon's Introduction to Biochemistry. Edited by W. J. E. Jessop, M.D., M.Sc., F.R.C.P.I., D.Ph., F.R.I.C.; fourth edition, 1961. London: William Heinemann Medical Books Limited. 8¾" × 6", pp. 480 with figures. Price: 30s. net (English).

EXAMINATION of this volume reveals that Dr. Jessop has maintained the characteristics of the earlier editions, and that extensive revision of the material in the previous edition has been effected. As with the earlier editions, a good deal of space is devoted to showing the student who has a limited knowledge of chemistry the variety of compounds present in various cells, and at the same time indicating the significance of these compounds. The orientation here is largely towards mammals.

The second portion of this book, called "Dynamic Biochemistry", leaves something to be desired. In the chapter "Metabolism of Carbohydrates", glycolysis and the hexose monophosphate shunt are dealt with, and the energy available from glycolysis is briefly discussed. No mention is made of the citric-acid cycle here, with its contribution to the energy obtained by the cell. "Tissue Respiration" is the chapter in which one finds a discussion of the citric-acid cycle. The cycle is said to be "concerned chiefly with the disposal of the end products of the glycolytic process and the lipo-elastic spiral", and little attention is paid to the quantity of energy available from this disposal. No discussion of the citric-acid cycle as a source of intermediates for biosynthesis is undertaken. Further, in the chapter "Protein Metabolism", the only aspect which appears to be dynamic is the transamination of amino acids. Proteins and the other components of the nitrogen pool are not considered in a dynamic sense.

We should, therefore, prefer to see the two parts of this book entitled "Part I—Biological Chemistry", and "Part II—Introduction to Biochemistry". Within the limitations outlined, this book is reasonably priced and usable.

General Zoological Microtechniques. By Frances M. Weesner; 1960. Baltimore: The Williams & Wilkins Company. 9" × 6", pp. 240. Price: 57s. 9d.

PROFICIENCY in the refined methods of microtechnique is acquired only by experience, but a basic series of simple, reliable methods can be easily mastered with practice. These are portrayed in Miss Weesner's book under the general headings of use of the microscope, fixation, preparation of stained and unstained whole mounts and preparation of stained serial sections. The book is intended primarily as a guide for students following a course in microtechnique,

and much attention is paid to general technical principles, detailed methods being interpolated as a series of well-chosen laboratory exercises on a wide range of vertebrate and invertebrate material. The advanced student or research worker using this book as a source for a particular method must bear with an excess of verbiage, but will find many useful items of technical common sense stemming from the author's long familiarity with her subject.

A major criticism of the book as a student text is the absence of discussion of the chemical basis of fixation and staining. Although the reasons for the success of many methods are obscure, a student sufficiently advanced to employ, for example, coal-tar dyes, should be made familiar with the rudiments of dye chemistry.

A number of minor omissions can also be noted. The employment of sodium iodate as a ripening and preserving agent in hæmatoxylin stains seems to have escaped the author. There is no mention of Mayer's hæmalum, a particularly useful stain for sectioned material. The methyl benzoate-benzene method of transfer from alcohol to paraffin, with its advantages in minimizing hardening, and the elegant Peterfi celloidin double-embedding technique are also neglected. However, Miss Weesner's book is to be commended as a student text, and will be welcomed by all teachers of zoological and medical microtechnique as a substitute for dog-eared, roneoed practical schedules. Its one disadvantage for laboratory use is its loose binding; when will publishers learn that to withstand student handling a book has to be tough?

Clinical Pharmacology. By D. R. Laurence, M.D., M.R.C.P., and R. Moulton, M.B.; 1960. London: J. & A. Churchill Ltd. 8" × 5¼", pp. 498. Price: 32s.

THIS new textbook is intended, so the preface states, for students in their clinical years and also for newly-qualified practitioners. However, any textbook of drug therapy perforce has a much wider following than this, since it often presents the only up-to-date account of new drugs with the exception of the manufacturers' literature, which is inevitably compiled through rose-tinted spectacles. The busy practitioner looks to books of this kind to give him the essence of new developments, and hence the stupendous object of complete coverage of subject matter must be the goal of the authors.

In 29 chapters this book gives a good basic account of the pharmacology of our vast collection of drugs, dealing first with the type specimens of each group and then filling in the details with the more recent additions. The book opens with an interesting section on drug therapy, placebos, clinical trials, the development of new drugs and other general topics, and a great deal of wisdom is packed into these 20 pages. The authors continue then to take the reader systematically through the established groups of drugs, ending with further general consideration of pharmacology, such as the dosage, fate and distribution of drugs. Then follow three very useful appendices dealing with weights and measures, the dosage of antimalarial drugs and surgery in diabetic patients.

An excellent feature of this book is its essentially practical and yet thoroughly readable nature. It is sprinkled with quotations and anecdotes which make each chapter attractive as well as factual, and few readers will fail to be attracted by this happy balance of style. There is a list of selected references at the end of each chapter which gives the authors' suggestions for further reading. Wherever there are many similar drugs in a group, tables are given which compare them very simply in respect of dosages, methods of use and other relevant details. This is a very good feature, which saves a great deal of hunting through the text to select the drug of choice for a particular case.

This book can be thoroughly recommended to any practising physician, newly qualified or otherwise, who cannot fail to find in it much worth-while information interestingly presented.

A Short Textbook of Hæmatology. By R. B. Thompson; 1961. London: Pitman Medical Publishing Co. Ltd. 8¼" × 5¼", pp. 306. Price: 47s. 3d. (English).

THE author has attempted to produce a synopsis of hæmatology which is a "readable account of disorders of the blood and blood-forming organs". In an era of rapidly expanding knowledge in this branch of medicine this is a difficult task; but the author has succeeded in presenting, from a clinical viewpoint, a clear and balanced account of this field.

As is stated in the preface, morphology and techniques are excluded, because they have become separate fields of specialized knowledge and experience. However, there are two plates of colour drawings of abnormal cell types, which, although good reproductions, do not aid the presentation of the somewhat inadequate morphological and hæmatological data on each disorder.

The book is nice to handle and the format of the contents is clear; in the many good tables of ætiology and of differential diagnosis and in the high-quality, carefully selected graphs, one perceives a realistic approach to the subject. The author's prose, although a little diffuse in some sections, is easy to read.

The introductory chapters on erythrocytes, leucocytes and blood groups are followed by chapters on each of the major hæmatological disorders. The space allocation bears a reasonable relationship to common clinical problems. Certain unsatisfactory terminology and data are retained without qualification in a book of this type—for example, absolute indices, and an illustration of a Price-Jones curve.

The chapter on hæmolytic anæmias could set out more clearly the problems associated with the diagnosis of enzymatic defects in red cells.

There is a great lack of detail on the diagnostic data, obtained by isotopic tracer studies, in megaloblastic and hæmolytic anæmia and in determination of blood volume. A notable omission is that of currently used radioactive vitamin B_{12} absorption tests.

Each chapter lists only a few general references, and in the chapters on megaloblastic anæmia, no reference is made to the publications of Mollin, Chanarin *et alii*.

The book should prove of value to general physicians, resident medical officers and students who want a good synopsis of clinical hæmatology which is a little more advanced than that found in general medical textbooks.

Motor Examination of Peripheral Nerve Injuries. By Y. T. Oester, M.S., Ph.D., M.D., and J. H. Mayer, jun., M.D., F.A.C.S.; 1960. Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications Ltd. 10" × 6¾", pp. 88, with many illustrations. Price: 44s. (English).

THIS small book has been published, as is pointed out in its introduction, to assist the relatively inexperienced examiner in evaluation of peripheral nerve motor function. Its 89 pages contain no less than 100 photographs showing a number of normal muscular actions as well as the abnormal actions resulting from peripheral nerve damage. It is, in fact, a book of illustrations, in which the written subject matter is little more than an expanded legend to explain the photographs. A number of the latter are somewhat disappointing, as they lack perspective, and the necessary detail is not shown up by the proper use of light and shadow, with the result that the reader must spend time and effort over several of them to ascertain the details. Nevertheless, there are a number of useful features, particularly with reference to substitution and trick movements which occur as a confusing factor with this type of injury. As a reference guide to peripheral nerve injury, it falls short in dealing with the concomitant sensory changes only by means of schematic diagrams.

This book is well printed on good paper, and despite the foregoing criticisms, the illustrations are ably reproduced.

The Psychological Care of the Child in Hospital. By Agatha H. Bowley, Ph.D., F.B.Psy.S.; 1961. Edinburgh and London: E. & S. Livingstone Ltd. 7¾" × 5", pp. 46, with illustrations. Price: 4s. 6d. (English).

THE hospitalization of children has been the subject of much discussion in both medical and lay Press in recent years. The Platt report (1959) on the welfare of children in hospital covered many aspects of child care, and made it clear that the care of the sick child requires special training, experience and understanding of children. Psychiatrists have been assuring us for years that the child under seven is at a very vulnerable stage of existence, and illness and the care he receives during this illness may leave indelible marks on his personality. Unfortunately some pædiatricians have been carried away by the special needs of children, and are loudly insisting that children should be cared for only in children's hospitals, or perhaps, as a very poor second best, in a pædiatric section of a general hospital. They often completely fail to face the problem that this huge country does not lend itself to that type of

service, and that either children must be taken hundreds of miles for minor illnesses, or there must be much more extensive training of medical and nursing staff in child care.

Few doctors will question that there are many medical and surgical conditions of children that require highly specialized care, such as that provided by teaching hospitals and children's hospitals, although there are a few general physicians who will maintain that disease is disease no matter what size the body. However, the vast majority of sick children will be cared for at home or in children's wards throughout the country, and it is very important that those who care for them become familiar with the modern approach to the child in hospital.

The present excellent booklet, of negligible price, should be acquired by every nurse and doctor and by most mothers who care for sick children. It is a soft-covered booklet of 47 easy-to-read pages, and deals with parents and nurses and their reactions as well as with the child's reaction to hospital and illness. There is a chapter on "Play, Occupations and Educational Activities", and an appendix on "The Care of the Handicapped Child". If every hospital in Australia had copies of this booklet together with the Platt report for its doctors and nurses to read, there would be a noticeable improvement in the handling of children in hospital.

The Day Hospital Movement in Great Britain: An Analysis and Description of Sixty-Five Day Hospitals and Day Centres with Special Reference to Psychiatric and Geriatric Day Hospitals Visited in 1958-9. By James Farndale, B.Com., F.H.A., M.R.S.H.; 1960. Oxford, London, New York, Paris: Pergamon Press. 9" × 6", pp. 448, with illustrations. Price: 84s. net (English).

THIS book is the report on a year's study by the author of the "day hospitals" of Great Britain. The author's experience in the administration of psychiatric units makes him well qualified to undertake a critical appraisal of this relatively recent development in the hospital field.

The book is set out in two parts. Part II, which comprises half the volume, is devoted to a detailed inventory of the 65 hospitals visited by the author during his study. Part I contains the history and background of the movement, and a detailed analysis of the various administrative problems associated with this type of hospital. The special aspects of the various types of day hospital, particularly psychiatric and geriatric, are considered.

At the end of Part I the conclusions drawn from the study are presented. It is interesting to note that no immediate financial benefit to the hospital system is anticipated from the introduction of day hospitals, as they cater for groups of the population which are at present inadequately covered. The system would appear to be well worth consideration as a possible supplement to psychiatric and geriatric hospital facilities in this country. Up to the present it appears to have been applied only on a very limited scale.

Mr. Farndale's book is attractively presented, and contains a number of illustrations depicting various activities of day hospitals. A useful bibliography is included. The book should be a valuable reference to those engaged in planning the future of our hospital system, particularly in the sphere of mental health and geriatrics.

A Synopsis of Physiology. Edited by C. C. N. Vass, M.Sc., Ph.D., M.B., Ch.B.; fifth edition, 1961. Bristol: John Wright & Sons Ltd. 7¼" × 4¾", pp. 354. Price: 30s. (English).

IT may be argued that there is no place at all for such books as this, since the compression of a subject so rich in details and complex interactions, and so poor in basic principles, can at best lead only to an erratic understanding and at worst to complete confusion. The originators are obviously aware of this danger, and the preface to the first edition (1927) makes it clear that this book is designed only as an aid to revision, and is for those already well informed in the subject through the study of major textbooks or from lectures. There is, in fact, little danger that anyone would try, against this advice, to use it as a textbook, since its tabular form makes forbidding reading, and some sections without diagrams, for example that on the ear, would be virtually unintelligible to the uninformed. There are altogether only 21 rather sketchy figures, including, surprisingly, a full-page plate illustrating urinary deposits, and a very misleading diagram of the events of the cardiac cycle, in which the pressure-tracings have been displaced for convenience of display, but this is not stated.

The new edition does appear, as its preface indicates, to have been carefully brought up to date, and contains a large and useful collection of quantitative data, some of which are not to be found in the standard text books; it may thus be a handy adjunct to their study. One imagines that it will find most use among candidates for fellowship and membership examinations rather than among medical students, since the latter should already possess adequate lecture notes of their own. To the former it can confidently be recommended as a useful set of headings and notes, about which their reading and study may be organized. The book is conveniently portable and has an extensive index; however, although authors are cited from time to time, there is no bibliography.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Year Book of Endocrinology", 1960-1961 Year Book Series, edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P.; 1961. Chicago: Year Book Medical Publishers. Sydney: W. Ramsay Surgical (N.S.W.) Pty. Ltd. 7¾" × 5¼", pp. 408 with illustrations. Price: 88s.

Ciba Foundation Symposium on "The Nature of Sleep", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., M.R.C.P., and Maeve O'Connor, B.A.; 1961. London: J. & A. Churchill Ltd. 8" × 5½", pp. 416, with illustrations. Price: 50s. net (English).

"An Introduction to Clinical Medicine", by R. R. H. Lovell, M.D. (Lond.), F.R.C.P., F.R.A.C.P., and A. E. Doyle, M.D. (Lond.), M.R.C.P., M.R.A.C.P.; 1961. London: Edward Arnold (Publishers) Ltd. 8¾" × 5¼", pp. 116. Price: 12s. 6d. (English).

"Chemistry and Therapy of Chronic Cardiovascular Disease", by Richard J. Jones, A.B., M.A., M.D., and Louis Cohen, B.S., M.D.; 1961. Springfield: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 6", pp. 200 with illustrations. Price: 60s. (English).

"Hormones in Blood", edited by C. H. Gray and A. L. Bacharach; 1961. London, New York: Academic Press. 9" × 6", pp. 656. Price: $20.00.

"The Year Book of Pathology and Clinical Pathology", 1960-1961 Year Book Series, edited by W. B. Wartman, B.S., M.D.; 1961. Chicago: Year Book Medical Publishers; Sydney: W. Ramsay Surgical (N.S.W.) Pty. Ltd. 7¾" × 5¼", pp. 454 with illustrations. Price: 99s.

"Programme Development in the Mental Health Field", tenth report of the Expert Committee on Mental Health; World Health Organization Technical Report Series, No. 223; 1961. Geneva: World Health Organization. 9¼" × 6¼", pp. 55. Price: 3s. 6d.

"Your Heart: A Handbook for Laymen", by H. M. Marvin, M.D.; 1960. London and Melbourne: Hodder & Stoughton. 7¾" × 5¼", pp. 304. Price: 31s. 9d.

"The Surgery of Trauma in the Tropics", by R. L. Batten, B.A. (Cantab.), F.R.C.S., with a chapter on anæsthetics by J. V. Farman, M.B., B.S. (Lond.), F.F.A.R.C.S.; 1961. London: Edward Arnold (Publishers) Ltd. 8¼" × 5¼", pp. 234 with illustrations. Price: 35s. net.

"The Vesico-Vaginal Fistula", by J. Chassar Moir, C.B.E., M.D., M.A., D.M., F.R.C.S. Ed., F.R.C.O.G.; 1961. London: Baillière, Tindall and Cox. 8¾" × 5¾", pp. 152 with illustrations. Price: 40s. net (English).

"The Patient's Attitude to Nursing Care", by Anne McGhee, A.M.I.A., with forewords by James Drever, M.A., F.R.S.E., and J. H. F. Brotherston, M.A., M.D., F.R.C.P.E., F.R.S.E.; 1961. Edinburgh, London: E. & S. Livingstone Ltd. 8¼" × 5¼", pp. 96. Price: 10s. 6d.

"Your Baby and Your Figure: Post-Natal Exercises", by the Obstetric Association of Chartered Physiotherapists, with a foreword by Professor Norman Morris; 1961. Edinburgh, London: E. & S. Livingstone Ltd. 7¼" × 4¾", pp. 12 with illustrations. Price: 2s. net (English).

"Essays on the First Hundred Years of Anæsthesia", by W. Stanley Sykes, M.B.E., M.B., B.Chir. (Cantab.), D.A.; Volume II; 1961. Edinburgh: E. & S. Livingstone Ltd. 8¾" × 6¼", pp. 188 with illustrations. Price: 30s. net (English).

"Surgery for Students of Nursing", by John Cairney, C.M.G., D.Sc., M.D., F.R.A.C.S., and J. Cairney, B.Sc., M.B., Ch.B., M.C.R.A.; fourth edition; 1961. Christchurch: N. M. Peryer Limited. 8½" × 5½", pp. 472 with illustrations. Price: 47s. 6d.

The Medical Journal of Australia

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THE VICTORIOUS INVALID.

EVERY PRACTITIONER numbers among his patients some at whose unconquerable spirit he marvels. They face discomfort and danger with cheerful courage. They overcome disabilities with ingenuity and resourcefulness. Their active minds use constructively the enforced physical idleness. In the highest category come those invalids who not only have met their adversity bravely but have actually fashioned from it an instrument of victory. They merit the description of Saint Paul as "more than conquerors".

The literature is rich with examples, of which only a few can be noted here. In 1948, *The Lancet* published a series under the heading "Disabilities", beginning with a girl of 23 years who learned to live triumphantly with traumatic paraplegia.[1] Our title is borrowed from Lane Crauford, who was laid aside for years with a tuberculous spine. In his book[2] "The Idle Hours of a Victorious Invalid", he records the incidents of his illness, offers his views on related themes and discusses the 55 doctors who had at one time or another attended him.

John Hadfield tells how he fell sick with a treacherous illness which confronted him with intimations of mortality and enforced on him time and leisure to reflect. After his first anxiety had subsided, he realized that his dominant impulse was one of intensified delight in the joys of life, in the inexhaustible variety of pleasures which his senses could still enjoy. He concluded that what made life so abundantly, so triumphantly, worth living was beauty. Out of these meditations came his "Book of Beauty", wherein each example of beauty of picture is matched with one of beauty of prose or poem.[3]

Louis Braille, born near Paris in 1809, the son of a harness maker, blinded himself at the age of three by playing with his father's awl.[4] He became first a pupil, later an instructor, in the National Institution for Blind Children in Paris. At the age of 16, he became absorbed with the idea of inventing a really efficient system of reading and writing for the blind. The awl, the very object that had destroyed his sight, was the instrument he used for making the letters. When he was 20, he published the Braille alphabet. Out of his own blindness, he brought light into thousands of darkened lives.

It was also at the age of three years that troubles began for Hideyo Noguchi, the son of a Japanese peasant.[5] He fell into the fire, and the fingers of his left hand were so badly burned that only twisted and scarred stumps remained. That disaster made him a distinguished scientist. The deformed hand prevented his becoming a farmer like his forebears, and the mocking of his schoolmates drove him to surpass them in his studies. A surgeon made something useful out of the stump, and that decided him to become a doctor. But when he graduated, he could not practise because of his hand. Thus was he directed into scientific medicine to become world-famous for his discoveries.

In 1873, Edward Trudeau, a young New York physician, became very ill with tuberculosis and was told he had six months to live.[6] He decided to spend his last days in a place in the Adirondacks that he loved. There, very gradually, his health improved. From his own agonizing experience, he evolved his method of treatment, which was to bring hope and health to hundreds of consumptives. His sanatorium at Saranac Lake became the model for American sanatoria. Here among others Robert Louis Stevenson, also a victorious invalid, spent the summer of 1887 while he wrote part of "The Master of Ballantrae". Actually Trudeau lived to the age of 67, and in later life was able to say: "The struggle with tuberculosis has brought me experiences and left me recollections which I would not have known otherwise, and which I would not exchange for the wealth of the Indies."

As a young surgeon, Eric Jeffrey was attacked by rheumatoid arthritis, and its unrelenting progress confined him to bed from the age of 30.[7] For the next 15 years, his bedside was a focus of activity. He studied medicine, general literature and classics, and systematically planned his reading for months ahead. He reviewed books for this Journal, prepared and delivered broadcast talks on medical subjects, corrected examination papers in physiology, and wrote the Jackson Lecture "The Ancient Romans Through Medical Eyes" for the Queensland Branch of the British Medical Association.[8] He suffered much from repeated exacerbations, but never complained. Those who came to console found themselves in a stimulating world of new interests. "His bedroom became a club in which good fellowship and intellectual battles were harmoniously linked" and the medical affairs of Tasmania thrashed out.

Who has ever described his personal experience with rheumatoid arthritis so vividly or so movingly as Reg Ellery?[9] It began in 1937 with transient attacks of metatarsal arthritis. For several years pain and swelling came and went. Remissions brought hope, recurrences foreboding. Joint after joint was attacked. In spite of

[1] *Lancet*, 1948, 1: 534.
[2] Chapman and Hall, London, 1925.
[3] Hulton Press, London, 1952.
[4] *Encyclopædia Britannica*, 1947, 3: 722.
[5] Williams, H., "The Conquest of Fear", Jonathan Cape, London, 1952.
[6] Williams, H., "Doctors Differ", Jonathan Cape, London, 1946.
[7] MED. J. AUST., 1935, 1: 69.
[8] MED. J. AUST., 1934, 2: 439.
[9] "The Cow Jumped over the Moon", Cheshire, Melbourne, 1956.

increasing distress and deformity, he continued with his practice. "The stricken patient puts his crippled hand to the plough, while he drags his arthritic feet painfully through the vineyard. . . . Pain spread over the day and rigidly disquieted the night. . . . A long drawn-out narrative of pain and disability, the slow torture of the rack periodically applied, where the joints are gradually destroyed but the spirit may remain unquenched. . . . The body a tangled mass of rusty pivots and broken hinges." Uveitis threatened to add blindness to immobility, but a gift of cortisone—then unavailable in Australia—restored his sight. In 1955 as the shadows closed round, he completed a review of his life, concealing a literary masterpiece under the title of a nursery rhyme.[9] He records how books were his first love and words never lost their fascination, how music came as a balm to his torn spirit. He racily recounts his unconventional adventurings and follows his imagination into enchanting by-paths. He recalls how the inarticulate cry of mentally defective children for help woke in him an evangelistic zeal for psychiatric reform, and finds the deepest satisfaction in memories of the gratitude of those he had liberated from emotional conflicts and ruinous fantasies.

Our examples of the unconquerable spirit have been drawn mainly from doctor invalids, but space must be found for W. E. Henley.[10] In 1860, at the age of 21, he had already lost one foot with tuberculous osteitis, and surgeons at Gloucester had told him that if his life was to be saved he must lose the other. But he had heard that Professor Lister of Edinburgh, with his new methods, often saved limbs and, ill and in pain, he struggled thither. For nearly two years he lay in the Infirmary—"this place of wrath and tears"—while, under Lister's care, both foot and life were saved. In his "Hospital Rhymes", Henley portrayed the personalities of the hospital and fiercely described the sufferings and frustrations of the patients. Lamed for life, penniless and with his future uncertain, he could write his immortal "Invictus". His vibrant affirmation

> I am the master of my fate:
> I am the captain of my soul

challenges every self-pitying, resentful or dispirited patient.

Let the last word be with Marjorie Lawrence, for in revealing the secret of her own victory she speaks for many. As Brünnhilde she had made history by galloping her horse onto the stage of the New York Metropolitan Opera House. In that dark hour when she was paralysed by poliomyelitis and her singing career was apparently ended, there was one thought[11] that became her mainstay:

> The Lord is my light and my salvation;
> Whom shall I fear?
> The Lord is the strength of my life;
> Of whom shall I be afraid?

That, and the never-failing encouragement of her husband, sustained her through the long and wearisome months of treatment, until eventually she returned to the stage and, although it was from a wheel chair, sang again to the public.

[10] MacCarthy, Mary, "Handicaps: Six Studies", Longmans, Green and Co., London, 1936.
[11] Psalm 27 : 1.

Comments and Abstracts.

THE STANDARDS OF SURGICAL MATERIALS.

THE many dangers attending implants of all types used as replacements in surgery were recently discussed by the Royal Australasian College of Surgeons when it became clear that many disquieting reactions were occurring. Elsewhere in this issue (see page 784) E. A. Allcock reports the failure in different ways of two internal splints. And so the story of the surgical introduction of foreign materials into the body goes on—a story that needs constant review.

Internal fixation of fractures is necessary only until the fracture has healed. From this point onwards the splint itself is not under stress, and provided it does not impede the movement of overlying muscle and is non-irritant to the tissues, it will cause no reactions. Prostheses on the other hand are quite different, for they must function satisfactorily for the rest of the patient's life. Great ingenuity and an adventurous spirit have been brought to bear in the design of these prostheses, and the number of materials used has become legion. Many plastic materials available have some tempting cherry to offer the surgeon. The first bite of most of these may have appeared promising, but the second has, with few exceptions, proved exceedingly disappointing. Such an exception, which still is in use 20 years after its introduction, is the Smith-Petersen cup for arthroplasty of the hip. Of the plastic materials methacrylate, introduced a decade ago, has been found inadequate, having destroyed many hips before its shortcomings became obvious. Recently we have read with grateful thanks the bold self-examination of "The Obituary to Ivalon" by Fitch, Denman and Waldon.[1] In the plastics field, self-lubricating prostheses for the hip are coming forward, and polypropylene appears to have characters which will displace both "Teflon" and "Terylene" from the field of prosthetic surgery.

For metallic implants the stainless steels have long held pride of place, but the cobalt chrome alloys appear to be superior as implants though their brittle nature restricts their use. It may well be that one of the results of the enormous advances that have occurred in metallurgical sciences to make modern rocketry feasible will be the introduction of many new alloys which are inert in tissues. Some of the new titanium-tungsten-molybdenum-vanadium alloys answer the established criteria, for they are tough and resilient and not subject to fatigue hardening.

These new materials are likely to be used clinically before we know much, if any, of their true biological nature. At present we have no means of checking whether the materials are suitable for use as implants, apart from the bland assurance of the manufacturer based on incomplete preliminary reports of very short follow-up studies. We have the medical directory to allow ready cross-check of any individual's credentials. We have the pharmacopœia to give us some idea of the actions of various drugs and to set a standard for their preparation. According to A. R. Docking[2] the dentists both in America and in Australia have a very efficient system for monitoring all the materials they use in order to keep them up to minimal standards. These bureaux have been in existence for 25 years and more. What of the field of surgery?

Some years ago J. S. Barr in two very thorough reports[3,4] pointed to the need to establish a standards bureau in the United States, a move which brought much opposition from certain quarters. In the more recent past (August, 1959) the British Standards Institution has produced a number of recommendations for metallic surgical implants, drills and screwdrivers. At a time when new materials are

[1] *Arch. Surg.*, 1960, 81 : 824.
[2] *Aust. dent. J.*, 1960, 5 : S248.
[3] *J. Bone Jt Surg.*, 1952, 34A : 249.
[4] *Ibid.*, 1954, 36A : 422.

confusing in their variety it is unthinkable that the untold misery raised by the uncritical acceptance of the Judet prosthesis should be repeated. A standards laboratory to make systematic investigations of all implant materials is now essential. These materials are not like simple drugs which are safe because they are so rapidly eliminated; prostheses are put in "for keeps". It is essential that they should fulfil the purpose for which they were designed. An essential preliminary for this is a series of standards which may be used as minimal requirements.

MIDWIFERY IN A PRIMITIVE COMMUNITY.

ONE wonders whether those who casually handle and discard the placenta in our sophisticated midwifery hospitals would not hold it a little longer in their hands and appraise it a little more thoughtfully if they knew the value placed upon it by more primitive peoples. A letter from a district nurse in Lahad-Datu, British North Borneo, which has been made available to us, reveals some fascinating details of its fate in that part of the world. The Suluks keep it and read the baby's future from it. The land Bajaus bury it beneath the steps of the house—on the left hand side in the case of a female birth and on the right in the case of a male—while the sea Bajaus give it a ceremonial burial at sea. The Cocos Islanders keep it for three days in a fancy tin, to which they add nails to keep away evil spirits and a ten-cent piece to ensure the child's material welfare throughout his life. Magical powers are ascribed to the cord, of which about a foot is dried after separation and worn around the neck in a bag for luck. Membranes are also used in this way, and sometimes a piece may be given to someone present at the birth.

Primitive obstetrics goes hand in hand with primitive superstition. Women are delivered fully clothed and wrapped in a sarong with a hole "the size of a sugar bowl" left for the head to come through. Nothing is done to control a speedy delivery, although leaves are applied to the vulva in case of a delayed one, and no interference with the cord is permitted until after separation of the placenta. The nurse records the case of a patient with a retained placenta of 21 hours' duration whose baby, covered with a cloth for warmth, was still lying with cord attached on the floor between her legs when the nurse arrived. When eventually the cord is cut, it is dressed with baked earth, and the placental portion is milked of blood, which is placed in the baby's mouth. Post-partum hæmorrhage is treated by tying pieces of string tightly around the patient's great toes.

The relative values placed upon baby and placenta seem actually to be reversed, since the baby is not even considered to be alive until it is six weeks old. At this stage its hair is cut in celebration, but a sick baby under six weeks of age receives no medical treatment at all; for if it is not yet alive, how can it die? A sick baby is not allowed to sleep; for if it does, the evil spirits will be able to enter its body. However, sometimes the parents will give a sick child to someone else in order to confuse the spirits and enable the child to get better. One can imagine the frustration and despair of a doctor confronted with an ailing child to whom the parents will not allow him to give an injection. One can imagine the difficulties of dealing with native workmen who will not drive a tractor in which a workmate has been killed until goats have been offered to it to induce the evil spirits to leave. It would probably benefit us all to reflect on the tyrannies of superstition from which our modern knowledge has delivered us, and perhaps we might even pity the placenta for the lowly place it has come to occupy in our society.

As an editorial postscript we should like to salute the writer of the letter referred to, for her courage and resourcefulness in bringing help to a strange people under conditions of extreme isolation—the only qualified doctor available comes on a visit once a month.

SHORTER ABSTRACTS.

MEDICINE.

TRANSSEPTAL CATHETERIZATION OF THE LEFT HEART: OBSERVATIONS IN 56 PATIENTS. R. T. Singleton and L. Scherlis. *Amer. Heart J.*, 1960, 60: 879-885 (December).

THE authors report on a new method of left heart catheterization, developed in the cardio-vascular laboratories of the National Institute of Health by J. Ross and others in 1958. This method of transseptal catheterization involves puncturing the interatrial septum through a catheter inserted into the right atrium via the saphenous vessel. Through this needle small plastic catheters can be introduced to explore the left heart; in each instance satisfactory tracings were recorded from the left atrium, and the left ventricle was entered in 71% of the cases in which entry was attempted. The authors outline the advantages of this method over other techniques of left heart catheterization.

CARDIAC OUTPUT IN SYSTEMIC ARTERIO-VENOUS FISTULAS COMPLICATED BY HEART FAILURE. G. M. Spurny and J. A. Pierce, *Amer. Heart J.*, 1961, 61: 21-24 (January).

THE authors state that congestive heart failure is recognized as a complication of systemic arterio-venous fistulæ although not commonly seen in clinical practice. The development of this complication is related to the size and duration of the fistula. The authors have studied the cardiac output in three patients with arterio-venous fistulæ during the period of congestive heart failure. Cardiac output was greater than normal in two of the patients, and less than normal in one patient seven days prior to death. After repair of the fistula the cardiac output tends to fall with control of congestive heart failure.

PERNICIOUS ANÆMIA AND POLYCYTHÆMIA VERA. I. Lind, *Acta med. scand.*, 1961, 169: 213-219 (February).

THE author describes the case of a patient with pernicious anæmia, who after treatment with liver extracts developed polycythæmia vera. This sequence of events, which has been recorded on numerous occasions, is thought not to indicate any ætiological connexion between the two diseases or to indicate that the hæmatinic factor may cause polycythæmia. The occurrence of the two diseases in the same patient is thought to be fortuitous, the remedy of the anæmia allowing a latent tendency to polycythæmia to become manifest. A *forme fruste* of polycythæmia may appear as a leucocytic proliferation despite the lack of the hæmatinic factor.

THE ELECTROCARDIOGRAM OF THE MOUNTAINEER AT HIGH ALTITUDE. F. Jackson and H. Davies, *Brit. Heart J.*, 1960, 22: 671-687 (November).

THE authors describe some of the investigations carried out during the British Sola Khumbu Expedition of 1959 and led by Mr. J. A. Emlyn-Jones which had as its objective the ascent of Ama Dablam, an unclimbed Himalayan peak 22,400 feet high, some ten miles south of Mount Everest. Electrocardiograms of 12 mountaineers were recorded at different altitudes up to 19,150 feet. Right axis deviation and *T* wave inversion in the right precordial leads were found in both European and Sherpa climbers and *S-T* segment and *T* wave changes in the left precordial leads in members of the European group. The authors conclude that much remains to be learned about the cardio-vascular response to high altitudes and that the reactions of the human body are complicated. The authors give various theories as to the possible causation of the electrocardiographic changes.

COMBINED ASPIRIN AND CORTISONE TREATMENT OF ACUTE RHEUMATIC FEVER. J. D. H. Slater, *Ann. rheum. Dis.*, 1961, 20: 173-178 (June).

THE author discusses combined aspirin and cortisone treatment of acute rheumatic fever. An evaluation is made of a controlled therapeutic trial in 28 young men, which was designed to assess the response to cortisone. The course of acute rheumatic fever was followed when high doses of salicylate were used, either alone or combined with cortisone. The patients were divided into two treatment groups; aspirin was given in divided doses totalling 1 to 1·5 grains per lb. body weight per day, and therapy was identical

except for the addition of cortisone to one group. The duration of fever, extent of joint involvement, and degree of elevation of the erythrocyte sedimentation rate were considerably reduced by giving cortisone in addition to aspirin.

A CONTROLLED TRIAL OF PHENYLBUTAZONE, OXYPHENBUTAZONE AND A PLACEBO IN THE TREATMENT OF RHEUMATIC ARTHRITIS. R. I. Meanock and E. Lewis-Faning, *Ann. rheum. Dis.*, 1961, 20: 161-172 (June).

IN this article the authors present the result of a controlled trial of phenylbutazone, oxyphenbutazone and a placebo in the treatment of rheumatic arthritis. The three forms of therapy were administered in succession to 60 patients with rheumatoid arthritis. Their mean age was 53 years and the average duration of the disease was five years. Each patient received each treatment for three weeks; assessments were recorded of grip, degree of pain, freedom of movement, functional capacity, and side effects. Results were analogous with that of other workers, that phenylbutazone ("Butazolidin") and oxyphenbutazone ("Tanderil") are both potentially effective in the symptomatic treatment of rheumatoid arthritis, and that both drugs give significantly better results than the placebo. No significant differences could be demonstrated between phenylbutazone and oxyphenbutazone, and any small differences obtained were not consistent. There were no significant differences between the percentages of side effects recorded from phenylbutazone, oxyphenbutazone and the placebo.

USE OF SPIRONOLACTONE IN RENAL EDEMA. R. T. Manning and F. C. Behrle, *J. Amer. med. Ass.*, 1961, 176: 769-771 (June 3).

THE authors discuss the use of spironolactone (an aldosterone antagonist) in renal œdema, its effectiveness and its association with hyperkalæmia. The clinical notes of six patients (four adults and two children) with œdema of renal origin are presented, in whom spironolactone was administered either alone or in combination with other diuretics. Three of the adults had diabetic nephropathy and one apparently had a sulpha-induced nephritis, whereas the two children appeared to have lipid nephrosis without evidence of glomerulo-nephritis. Spironolactone appears to to be an effective addition to therapy of œdema of renal origin. The major undesired effect is the occurrence of hyperkalæmia in those individuals with severe renal dysfunction as measured by elevated blood urea nitrogen. It is pointed out that hyperkalæmia may develop during spironolactone administration, despite concurrent administration of chlorothiazide, which is a known kaluretic agent. At present, steroid therapy undoubtedly offers the most benefit to children afflicted with the nephrotic syndrome, but initial experience with spironolactone suggests that it may serve as a valuable adjunct to steroid therapy, particularly when there is refractory œdema. In this series spironolactone was administered orally in doses of 200 to 400 mg. daily. Three of these patients developed hyperkalæmia during spironolactone administration and each had severe impairment of renal function. No other side effects were noted, and the drug appears to have a relatively wide margin of safety provided close observation of potassium blood level is maintained in patients with impairment of renal function.

EXCESS MORTALITY ASSOCIATED WITH EPIDEMIC INFLUENZA. T. C. Eickhoff *et alii*, *J. Amer. med. Ass.*, 1961, 176: 776-782 (June 3).

THE authors present some observations on the excess mortality associated with epidemic influenza. Statistical evidence shows that 80,000 deaths are estimated to have been caused by Asian influenza during the two epidemics that have occurred in the U.S.A. since 1957. About 40,000 of these deaths occurred during the first epidemic of 1957-1958, and 27,000 more excess deaths occurred during the second epidemic in 1960. The relationship of age and certain chronic disease states to the excess mortality associated with these epidemics of influenza was investigated. Over two-thirds of the excess mortality occurred in persons aged 65 years and over. Excess influenza-associated mortality was demonstrated in persons with cardio-vascular-renal disease, certain broncho-pulmonary diseases, and diabetes mellitus. Mortality data as well as clinical studies confirmed a strong association of rheumatic heart disease with influenza-associated deaths. The increased risk of influenza-associated death in pregnancy was supported by clinical studies. Immunological protection through the routine use of influenza vaccine in such high-risk groups may be of great value in reducing the extent of influenza-associated excess mortality.

THE CHOLESTEROL-FATS, DIETS, AND ATHEROSCLEROSIS PROBLEM. G. A. Goldsmith, *J. Amer. med. Ass.*, 1961, 176: 783-790 (June 3).

THE author discusses cholesterol-fats and diet and the atherosclerosis problem. Current concepts of lipid metabolism which may be of importance in elucidating relationships of dietary fats and serum lipid concentrations to the atherosclerosis problem are reviewed. Many dietary substances and numerous other factors influence serum lipid levels. Cholesterol is only one of the serum lipids which may be related in some way to atherogenesis. Specific triglycerides, phospholipids, fatty acids, or the whole spectrum of beta lipoproteins may be of equal or greater importance. Atherosclerosis appears to be a disease of multiple ætiology, with many endogenous and exogenous factors playing a rôle in the development of lesions. A direct causal relationship between diet or serum lipid concentrations and atherosclerosis or coronary artery disease remains unproved. Obesity, hypertension, and hypercholesterolæmia are associated with a high incidence of coronary artery disease. It appears logical to attempt to reduce high concentrations of cholesterol and other lipids in serum, as an experimental therapeutic procedure. The author considers that investigations should be continued to determine the value of diets moderate in fat and relatively rich in poly-unsaturated fatty acids in the prevention or alleviation of coronary artery disease, and that the possible rôle of fats in the blood coagulation mechanism merits further study. He concludes that until more information is available, it is impossible to recommend the fatty acid mixture or the total amount of dietary fat which may be desirable for the maintenance of health.

DRUG-RELATED BLOOD DYSCRASIAS. C. M. Huguley *et alii*, *J. Amer. med. Ass.*, 1961, 177: 23-26 (July 8).

THE authors review the question of drug-related blood dyscrasias, based on reports of 448 cases received during the year 1960 by the registry of the Study Group on Blood Dyscrasias of the A.M.A. Council on Drugs. The findings show that it is incumbent upon the physician to maintain an awareness of the risk of blood dyscrasia associated with the use of certain drugs, and to use such drugs only when the potential benefits of administration considerably outweigh the relatively small risk of developing a blood dyscrasia. With the use of such drugs, it is important that hæmatological studies be made at intervals, and that the patient be warned to report immediately the development of fever, sore throat, weakness, pallor or a bleeding tendency. Also, it is important that the physician be aware that in the event of development of infection or easy bruising in a patient who has been taking such drugs, blood cell counts must be made immediately. If agranulocytosis, thrombocytopenia, or pancytopenia is present, further administration of the drug must be stopped.

PERCUTANEOUS DIAGNOSTIC PUNCTURE OF THE HEART AND GREAT VESSELS. A. Tybjaerg Hansen *et alii*, *Acta med. scand.*, 1961, 169: 273-282 (March).

THE authors describe a method for determining the pressure gradients across the ostia of the left side of the heart in patients with mitral and aortic valvular diseases. Suprasternal puncture of the left atrium is combined with direct percutaneous puncture of the left ventricle. The method has been followed in 149 patients and a successful examination was made in 121. Serious complications appeared in eight patients, mostly syncopal in nature, and one very sick patient died.

USE OF DIURETICS IN HYPERTENSION. R. W. Gifford, junior, *J. Amer. med. Ass.*, 1961, 170: 70-71 (July 8).

THE author discusses the use of diuretics in hypertension, and states that thiazide diuretics have a place in the treatment of most patients with hypertension in whom the condition is severe enough to require specific treatment. When hypertension is mild, a thiazide agent may be the only drug needed, but when more potent anti-hypertensive drugs are required, concomitant treatment with a thiazide diuretic will often enhance the effectiveness of the other agents, and so permit better control of blood pressure with

smaller doses and consequently fewer side effects. Symptomatic side effects produced by thiazide diuretics are infrequent and rarely require cessation of therapy. Supplements of potassium are not necessary for most patients receiving thiazide diuretics, provided they can be kept under surveillance. Renal insufficiency with moderate azotæmia and blood urea figures of between 50 and 100 mg. per 100 ml. is a relative contraindication to the use thiazide diuretics, and when azotæmia is profound with blood urea figures of more than 100 mg. per 100 ml., these agents should not be used except for short periods when the patient is under careful observation. The only absolute contraindication to their use is drug sensitivity, which is rare. The author is not convinced that any one of the thiazide preparations presently available is superior to the others. When one is poorly tolerated, is ineffective, or produces a sensitivity reaction, another can be tried.

A SOLVENT FOR KIDNEY STONES. M. Brozinski *et alii, German Medical Monthly*, 1961, 6: 105 (April).

THE authors have shown that a 2·5% solution of disodium ethylenediamine acetate ("Titriplex III"), buffered with 3% triethanolamine at a pH of 8·6, will completely dissolve all calcium-containing types of kidney stones, including those containing calcium oxalate. The solution is infused continuously through a double-channelled catheter, the tip of which has been placed in the renal pelvis. The infusion has been maintained without side effects over many weeks and the method has successfully dissolved quite large stones.

LONG-TERM TREATMENT OF CIRRHOSIS OF THE LIVER WITH PREDNISONE. B. Harvald and S. Madsen, *Acta med. scand.*, 1961, 169: 381-387 (April).

THESE authors found that prednisone was never helpful in the treatment of portal cirrhosis and seldom in the treatment of biliary cirrhosis. However, in cirrhosis following upon hepatitis, that is to say in patients whose blood exhibited strongly positive thymol turbidity, high serum transaminase activity and a high level of gammaglobulin, the results of treatment were always good, often excellent, provided that the treatment was continued for a year or more. Repeated observation of the transaminase activity was a good index of progress. The decrease in this activity is interpreted as resulting from a sealing of the liver-cell membranes and a prevention of cytolysis analogous to the prevention of hæmolysis by the steroid therapy of hæmolytic anæmia.

INCREASED SUSPENSION STABILITY OF BLOOD AFTER HEPARIN ADMINISTRATION. R. Berlin and H. Sandring, *Acta med. scand.*, 1961, 169: 369-372 (April).

SEEKING to explain the slight retardation of the blood sedimentation rate which occurs after the administration of small amounts of heparin to persons with accelerated blood sedimentation, the authors have proceeded from the observation of Fåhraeus that incubation of the serum for some hours lowers the sedimentation rate when red corpuscles are added to it again. They have found that this effect is increased by the injection of small amounts of heparin and can be demonstrated as early as a few minutes after the injection. The explanation was not conclusively proved, but the authors suggest that the heparin acts as a liberator or activator of an enzyme system, such as a lecithinase or lipoprotein lipase, either of which might cause erythrocytes to lose their biconcavity and diminish their tendency to form rouleaux.

EARLIER DIALYSIS AND ANABOLIC STEROIDS IN ACUTE RENAL FAILURE. C. R. Blagg and F. M. Parsons, *Amer. Heart J.*, 1961, 61: 287-291 (March).

IN an editorial comment the authors state that of those patients referred for treatment with the artificial kidney approximately 50% can be classed as surgical or traumatic, 30% as medical and 20% as obstetric cases. The symptoms of uræmia are due to the accumulated products of tissue breakdown and treatment should therefore be directed to: (i) the removal of these products by dialysis, and (ii) the reduction of protein metabolism. The trend at present is towards earlier and more frequent dialysis. This is unnecessary in patients with acute tubular necrosis who have a rate of rise of blood urea nitrogen of less than 30 mg. per 100 ml. per day. With adequate treatment, including the use of the artificial kidneys when necessary (either on clinical grounds or when the blood urea nitrogen reaches 200 mg. per 100 ml.), the rate of recovery among such patients is more than 90%. Among patients with a high rate of protein metabolism, in whom the increase in blood urea nitrogen reaches more than 30 mg. per 100 ml. per day, recovery occurs in only about 30% of cases. Acute renal failure in this group of patients is almost always a complication of surgical or accidental trauma. Theoretically the rate of protein breakdown can be reduced in three ways: (i) by high calorie diet without protein, taken by mouth; (ii) by 50% glucose solution administered by caval catheter; (iii) by anabolic steroids. The first two methods are of only limited value, the patient is often unable to take a high calorie diet, and the risks of caval transfusion (thrombosis, perforation of the vena cava, infection and immobilization) outweigh its usefulness as a routine measure in most of the patients. Recently several new steroids related to testosterone, with a greater anabolic/androgenic ratio, have been introduced; these include norethandrolone, methandrostenalone and norandrostenolone phenylpropionate. It has been shown that in patients with acute renal failure due to obstetric causes these steroids will reduce the rate of protein breakdown by 60% to 70%. The authors conclude that the new anabolic steroids should be used routinely in the treatment of all patients suffering from acute reversible renal failure.

SURVIVAL AND REHABILITATION IN CORONARY OCCLUSION. A. M. Master, *Amer. J. Cardiol.*, 1961, 7: 340-349 (March).

As part of a symposium on rehabilitation in cardiovascular disease the author discusses the problem of coronary occlusion. The importance of returning a patient to work is emphasized both for his morale and for his physical well-being. The author stresses that neither effort nor stress or strain of modern life precipitates a coronary occlusion. Coronary occlusion occurs with equal frequency in all classes of society and in all types of occupation, perhaps slightly less commonly in those employed in physically laborious work. Coronary occlusion appears to be the end result of an atherosclerotic process, independent of external influences. The author considers that increased emotional stress and strain of present day working and living does not tend to produce coronary artery occlusion, the highly-strung and the phlegmantic person are equally susceptible to it.

RECENT DEVELOPMENTS IN HYPERTENSIVE THERAPY. F. M. Smirk, *Amer. Heart J.*, 1961, 61: 272-281 (February).

THE author states that few patients should suffer persistent discomfort from the use of effective hypotensive therapy. Although diuretics alone or even in combination with rauwolfia alkaloids will not always reduce blood pressure much more than a placebo, there is in almost every case a distinct enhancement of the response to ganglion-blocking drugs and to the new sympatholytic drugs, bretylium and guanethidine. For severe cases the additional use of a ganglion-blocking drug such as mecamylamine or pempidine is of great value. In patients who have a peptic ulcer it may be unwise to use bretylium or guanethidine without such a ganglion-blocking drug to restrain gastric secretion.

INTRACARDIAC MYXOMAS AND THROMBI. C. W. Adams *et alii, Amer. J. Cardiol.*, 1961, 7: 176-187 (February).

THE authors state that approximately 550 primary tumours of the heart have been reported, and that the majority are myxomas, arising typically from the atrial septum in the region of the fossa ovalis. They are three times more frequent in the left atrium than in the right. The incidence appears to be greater in women than in men. Clinical manifestations usually arise from an obstruction of the mitral or tricuspid valve. Peripheral emboli and low cardiac output may complicate the clinical picture. The authors describe the clinical features and investigations of three such cases. With the advent of successful surgical methods for the removal of these tumours, early clinical recognition is important. Angiocardiography is the most important single diagnostic procedure for identification of intracavitary cardiac tumours, and should be undertaken whenever the possibility of such a lesion exists.

On The Periphery.

NEITHER HEADS NOR TAILS.[1]

WHEN Julius Cæsar set out to write an account of one of his campaigns, he not only set an example that has been assiduously followed, with varying success, by many army commanders since his day; he also conferred immortality upon a very pedestrian statement: "All Gaul is divided into three parts." I want to suggest for your drowsy consideration that he could, without sacrifice of truth, have written "all humanity is divided into three parts". There are those who seem to be the specially favoured of the gods, with gifts of mind and accomplishment far in excess of the average. Everything seems to come easily to them, and they leave the launching pad and rise to positions of eminence and distinction in their chosen work, occupation, trade, profession or calling (if any) with a speed and ease suggestive more of the lift than of the ladder. They are of tremendous value to the community, but are few in number. At the other end of the scale are those who never really succeed in anything, but just manage to eke out a very precarious existence which they all too frequently terminate by looking for a gas leak with a lighted match, or per medium of the gun that was not loaded. They are, alas, inevitable, but are also mercifully few in number. And in between is the vast majority of mankind, the ordinary average citizens, not by any means dumb, nor yet uncomfortably embarrassed by brilliant gifts. They achieve satisfying success and live happy and very useful lives.

The spotlight of publicity is constantly focused upon the members of the first group. Merciful obscurity encloses the second. All too often those in the third group are taken for granted. This is wrong, for they are the really important section of the community, the people without whom the bright boys could not achieve their aims or fulfil their destiny.

No claim for originality is made for this statement. It is a self-evident truth of experience. Consider for a moment. In the Services, the top brass may formulate the most efficient plans for spreading confusion and dismay in the ranks of the enemy; but they depend for their success upon the courage and tenacity of purpose of the "P.B.I.", the man in the ranks (or, should I say, "the space capsule"?). In business, the up-and-coming executive may spend hours in "maturing his felonious little plans", but they will never mature unless he can depend upon the loyalty and discretion of the man in the outer office. In the law, forensic triumph is credited to the barrister for his oratory and his subtle employment of *suggestio falsi* and even *suppressio veri*; but where would he be without the services of the much less spectacular solicitor who "mugs up" the case for him? In the church, the most inspired preacher is but "a voice crying in the wilderness" if he has not in his congregation people with vision to share his ideals and courage and determination to translate them into action—a sobering thought to all who look for the day when mankind shall learn to live together in peace and amity. The same holds true in medicine. Collins Street is ready to dazzle you with science at the drop of a hat; but where would the high-powered consultant be without the support and cooperation of that stout fellow, the general practitioner? He seems to possess the faculty of enlisting the loyalty and winning the affection and trust of the average citizen. As witness this poem which appeared many years ago in *The Speculum*. It was copied from *St. Bartholomew's Hospital Reports*, and is cast in the mould of Kipling's "Soldier and Sailor Too".

THE COUNTRY DOCTOR.

As I was going 'ome to bed thro' a muddy country lane,
I seen a man in an oilskin cap atrudgin' thro' the rain.
'E 'adn't a match, an' 'is pipe was out, and I sez to 'im — oo are you?
An' 'e sez, I'm a doctor, a country doctor, surgeon and midwife too.
Now 'e never gets paid for 'arf 'e does, and 'e does the work of two,
An' 'e isn't one of the gentlefolks, an' 'e isn't like me nor you.
E's a sort of bloomin' chameleotripe, surgeon and midwife too.

An' I seen 'im agen all over the ship, aplayin' all sorts of gags,
Like settin' a fractured collarbone with a couple of touch-line flags.
An' the parsons owe 'im money, for their wives give 'im work to do,
Tho' 'e's only a doctor, the country doctor, surgeon and midwife too.
And the Poor Law Board they sets on 'im an' tries tc dock 'is screw,
Tho' 'e 'as 'is bread and cheese to get, same as me nor you.
They think 'e's a bloomin' philantocrat, surgeon and midwife too.

An' I seen 'im agen with a knife and things, and the sweat was on 'is brow,
'E was tryin' to mend the guts of a bloke as 'ad spiked 'imself in a row,
It was late at night, and 'e 'adn't no light to see what 'e 'ad to do.
An' 'is pal was a doctor, a country doctor, surgeon and midwife too.
'E 'adn't got far with 'is little job; 'e wasn't but arf way thro',
When the bloke sets up an' asks for a drink, same as it might be you,
Ho! they ain't no speshul anæsthetutes, surgeon and midwife too.

But there wasn't a call to do as you done when you 'ad the gout in your toe,
An' you fetched 'im out in the dead of night, and 'e 'ad six miles to go.
For you've 'ad it before, and you'll 'ave it again, and you know just what to do;
You don't want the poor old country doc, dispenser and staff nurse too.
You pays! What! Yes, tuppence a week, and you're earning your thirty-two,
An' 'e 'as to subscribe to your football club, which you're too mean to do.
Becos 'e's a doctor, a country doctor, surgeon and midwife too.

Now I never believes in them specialist thieves as stammer an' grunt an' blow,
An'll watch you die with a winkin' eye for a 'undred pounds or so,
An' when it's "checks!" and oos turn next (which I 'opes it won't be you)
Let's stick to the doctor, the country doctor, surgeon and midwife too.
An' when you comes to the bar of Gawd, an' 'e sez "oo passed you through?"
For 'e 'ates peculiar people an' the Christian Science crew,
Just mention the doctor, the country doctor, surgeon and midwife too.
(Apologies to Kipling.)

—E.G.B.A.

My bias towards the general practitioner began early in life, for both my father and my elder brother were general practitioners. I held them both in high regard and deep affection. As I remember my father first, he was a genuine "horse-and-buggy doctor", though he soon graduated, by way of a series of fearsome and temperamental "bombs", to a reliable form of motor transport. Judged by the standards of today, he would be regarded as hopelessly out of date. To take but one example, his

[1] After-dinner speech delivered at a meeting of the Victorian Branch of the British Medical Association, Geelong, August 19, 1961.

apparatus for giving an anæsthetic consisted of a folded table-napkin and a drop-bottle of chloroform, which appears perfectly archaic in comparison with the mobile milk-bar of the present-day anæsthetist. But he gave a good anæsthetic. His pharmacopœia was doubtless limited, but he knew its constituent drugs intimately, the virtues peculiar to their various preparations both singly and in combination. He wrote prescriptions in full, in Latin, and scorned the proprietary preparation. I should dearly love to hear what he had to say to the present-day agent with his sales-talk of "broad spectrum" and "remote side effects". His surgery was, perforce, of the kitchen-table and emergency variety, and on the one occasion on which I assisted him, I found that my main duty was to frustrate the clinical curiosity of the local blow-fly; but the wound healed by first intention. But he knew his patients. How he did know them! He met then in the street, at church and at social gatherings of all sorts. He was in and out of their homes. He knew their anxieties and their cares, their strengths and their weaknesses, their heredity and background, which ones to jolly along, which ones to bully. The result was that, when they brought him their illnesses, he was able to take into account many factors other than those presented by the symptoms, and his treatment was the better and more efficient in consequence. One day we stopped at the outer gate of a farm, and father rummaged in the tool-kit and took out a large screw-driver, which he carried with him into the house. On being asked what therapeutic function this rather unusual appliance was designed to fulfil, he answered: "That man has T.B. and his wife *will* keep the window shut. Last time I was here I told her that, if ever again I found it shut, I would take it out of its sash. I just wanted to show her I meant what I said."

Father belonged to a generation that is gone, and the pattern of general practice has undergone changes since his day. But though the pattern may have changed, the general practitioner remains, both numerically and qualitatively, the backbone of the profession. He is also, to no small extent, the custodian of its good name. We are all proud, and justly so, of the high regard in which Australian graduates are held overseas. This is, doubtless, largely due to the disproportionate number of outstanding and brilliant men that this country has produced. But it also owes a great deal, a very great deal, to the reputation established during two wars and the years between by our ordinary run-of-the-mill graduates, our general practitioners—a reputation for all-round ability, for initiative in crisis, for capacity to cope with all manner of medical emergencies and difficulties.

I have no doubt that the general practitioner of today is, and the general practitioner of tomorrow will be, able to maintain that reputation. But there are some features of our present-day way of life that give one cause for thought; trends which may, if not checked, lessen the opportunities for the young graduate to develop those very qualities upon which that reputation is based. I mention only two.

The first is the rapidity and ease of transport. In time past it was the custom for the citizen to attend the nearest doctor when in need of medical attenion. This was particularly true in the country, and it meant that the local general practitioner was called upon to treat all manner of conditions. It was a stimulus and a challenge to him to keep up to date in his work, to use his brains, to develop his knowledge and enlarge and learn from his experience. If he had any capacity at all, it gave opportunity to him to develop that capacity to its very fullest extent and to become a very knowledgeable and useful citizen. He learned to form a sound opinion and act upon it, to decide which cases he was able, in good conscience, to handle himself, and which required to be referred to one who, by training and experience, was better fitted to deal with the problem. Moreover, he lived with his patients in a more static community, got to know them intimately and, because of this knowledge, was the better able to treat their complaints. They were, in very truth, his people and he their doctor. Today it requires but the turn of a switch and continued pressure of the right foot for a patient to travel miles away to another town or big centre. This not only weakens the much-to-be-desired doctor-patient relationship, but also lessens the opportunity given to the general practitioner to develop his powers to the full. It is not only to the cow that the grass through the fence looks the greenest, and this tendency has the bad effect of leading the patient to under-estimate the worth of the local man in comparison with the one to whom distance lends enchantment. It must also be acknowledged that the ease with which a difficult or worrying case can be shipped off to the nearest hospital encourages the potentially lazy doctor to develop his potentiality to the full and to his own deterioration. The second factor is far more serious. It arises from the rapid, almost explosive, advances which have been made in medical knowledge and surgical possibilities in the last decade or two—advances which have so extended the field of medicine that it is now well-nigh impossible for one man to have an adequate knowledge of all branches of the healing art. This has stimulated specialization to an almost alarming extent, and the young man, if he wants to be a real expert, is tempted to specialize too early. This basic difficulty is responsible for the growing practice of "group practice", in which each member has special qualifications for and experience in some particular branch, so that the group, as a unit, is competent to deal expertly with anything that comes along. But this meets the situation only in part. It does not lessen the fault of early specialization and can even aggravate it. If this tendency to early specialization is allowed to grow unhindered, may we not reach a stage at which the general practitioner is regarded as one who is fitted to deal with comparatively minor ailments only, having as his chief function the capacity to advise the patient which specialist he should consult? A bogy? Not altogether, I fear. One has too often, in reply to the question, "Have you already seen a doctor?", received the reply, "Only the local G.P.". That "only" is ominous, for it indicates a state of mind which regards the general practitioner as something less than a "real doctor".

It will be a sad day for our profession if that opinion gains ground. It will be catastrophic if it ever takes root in the mind of the medical student, so that he regards going into general practice as a confession of mediocrity instead of being, as it is, an opportunity of developing what powers he has to their fullest and best. Should that day ever threaten to dawn, it may not be out of place to present to each new graduate a florin or lesser coin, and to point out to him that, however regrettable the tail and however glamorous and magnificent the head, the real strength and value of the coin are in the metal between, that part which is "neither heads nor tails".

ARCHIE S. ANDERSON.

Melbourne.

Out of the Past.

COUNTRY LIFE AND LUNACY.[1]

[From the *Australasian Medical Gazette*, August 20, 1903.]

IN his annual report on lunacy in South Australia, Dr. Cleland, Superintendent of the Parkside Asylum, says: "By far the larger proportion of admissions to the asylum come from a section of the community whose daily avocations are of the nature of outdoor life and laborious employment. Theoretically, it has a hygienic sound to be out all day long in the sunshine and fresh air; practically, it is found to lead to premature decay when combined with working for a living, owing to the general exposure to wind and weather; so that, although to a townsman 50 years of age may not sound specially elderly, yet, under the circumstances mentioned, it is quite equal to 60 years of age under more favourable conditions. If, added to this, is the frequent

[1] From the original in the Mitchell Library, Sydney.

privation and mental anxiety arising from the uncertainty of obtaining employment, it is not surprising to find that there is but a very small surplus of vital energy, if any, to help build up the exhausted brain centres after the mental breakdown."

Special Correspondence.

PARIS LETTER: MAY-JUNE, 1961.

(FROM OUR SPECIAL CORRESPONDENT.)

Can Foot-and-Mouth Disease be Transmitted to Man?

FOOT-AND-MOUTH DISEASE, a disease which is particularly contagious and easily transmitted by inoculation, is caused by a very small virus, which was the first ultravirus discovered. This virus is not single; up to the present time seven different types are known, of which three are rampant in France. The hypothesis that foot-and-mouth disease can be transmitted to man is not new. A great number of single cases, without experimental verification, have been reported as following either ingestion of milk or contact with an infected animal. Professor J. Verge,[1] Director of the Central Laboratory of Veterinary Research at Alfort, had his attention drawn to the fact that new employees at the laboratory were often affected by a kind of "'flu" a few days after their first contact with an infected animal. On several occasions after this observation had been made, serological tests were carried out on the blood of the personnel working in the foot-and-mouth disease service—complement fixation, neutralization and cold agglutinins. All the results were negative. It is, therefore, necessary to be extremely cautious on the question of the origin of an aphthous infection. Foot-and-mouth disease can be proved only by inoculation of suspected material into susceptible animals (cattle or guinea-pigs), or by the discovery of specific antibodies in the serum of convalescent patients. It seems that the transmission of this disease from animals to man occurs infinitely less often than it is usually considered to do. Human aphthosis, described by Touraine, which has a number of similarities to foot-and-mouth disease, cannot be transmitted to cattle or guinea-pigs. It is certain that the development of virus isolation techniques will prove that other viruses than that of foot-and-mouth disease are the cause of most cases of human stomatitis.

The Limitations of the Sulphonamide-Biguanide Combination in the Treatment of Diabetes Mellitus.

Should the indications for oral treatment of diabetes mellitus be revised? The sulphonamides have been joined by the biguanides, and a combination of the two drugs is occasionally more effective than one or other used alone; but the effectiveness of this combination also has limitations. In contrast with some other workers, Roger Deuil, M. Magdelaine and P. Tartinville[2] do not think that the sulphonamide-biguanide combination provides sufficiently reliable control of juvenile diabetes, even when it is mild and in the early stages. In their opinion, insulin therapy is the best form of treatment for patients aged under 40 years and of less than normal weight, even if they have no family history of diabetes and no ketonuria. The combination of the two drugs may appear to reduce the diabetes, but these workers have not been able to produce in their patients a feeling of well-being and a return to normal weight. Oral treatment (a sulphonamide, biguanide or a combination of the two) is of value for patients aged more than 40 years whose weight is above normal, but often loses its value if treatment of obesity is carried to its conclusion. With the foregoing reservations, it is certain that a vast number of diabetics benefit from oral treatment. It protects them from the many complications brought about by hyperglycæmia. The advent of the guanidines has increased still more the number of patients who are able to dispense with insulin. Oral treatment requires regular supervision of the patient and must not interfere with strict dietary control; moreover, those patients who regain a normal weight and can thereafter do without non-insulin treatment are unhappily rare. In the treatment of young patients, the authors hold that insulin can never be entirely replaced; the sulphonamide-biguanidine combination has only a partial action, and its effect wears off fairly rapidly.

Polyuric Cirrhosis of the Liver.

It is a platitude to state that in cirrhosis of the liver the prognosis is written on the vessel containing the urine. Actually, normal excretion of urine still remains the best sign of clinical improvement. However, M. Bariety, A. Gajdos and P. Choubrac[3] have observed several cases of cirrhosis with ascites in which this opinion was wrong. The condition was a special clinical type of cirrhosis, characterized chiefly by polyuria, with the excretion of 2 litres of urine and pronounced reduction of sodium excretion. The polyuria appeared after a long clinical course with repeated attacks of ascites, at a time when all the clinical and biological signs pointed to cirrhosis with grave decompensation. Yet the polyuria did not prevent the rapid development of further ascites, and was in no sense a favourable prognostic sign, since the condition rapidly terminated fatally. It can thus be concluded that very poor urinary excretion of sodium is an observation that characterizes even more closely than oliguria, renal function in cirrhosis with ascites. Polyuria is in no sense a favourable prognostic sign.

Subperitoneoscopic Forceps Biopsy.

This method is not new; but recently the more extensive use of laparoscopy and the requirements of exact endoscopic diagnosis have led various workers to use again this forceps biopsy, which had been rather overlooked. Ch. Debray, J. Paolaggi, R. Laumonier and E. Martin[4] use bone-cutting forceps made according to the model of M. Royer, 2 mm. in diameter and with only one moving blade. This forceps is independent of the laparoscope; it is inserted into the abdomen at a different point of entrance from that of the latter. A disadvantage of this separation is the need for a second point of entrance, a second area of local anæsthesia and a second incision; but it has great advantages over Ruddock's forceps, which are incorporated in the laparoscope. It can reach inaccessible areas beyond the end of the endoscope, it does not hinder photographic and cinematographic work, and finally it permits the carrying out of manœuvres such as the removal of adhesions or the replacing of omental fringes, which would not have been possible if the forceps had been in one piece with the endoscope. This method of biopsy can be used to take specimens from both the liver and the peritoneum. When it is applied to the liver, it is particularly useful for the study of cortical structure (perihepatitis, diagnosis of the "candle-grease markings" of certain types of cancer). In its application to the peritoneum, it is the only technique of biopsy under observation that is available. It makes possible the quick and certain diagnosis of tuberculosis, of peritoneal carcinomatosis and of granulomatosis of the peritoneum.

Non-Specific Prostatitis.

This relatively rare disease, recently discovered, has been described by P. Aboulker, J. Roujeau, J. Chome and Cl. Rougement[5] as a prostatic disease of later life, chronically progressive, and often mistaken for malignant disease. It is due to a macrophagic reaction of the histiocytes to contact with the lipids of the gland cells from tissue necrosis and the dissemination of the products of secretion in the connective tissue. The interest of these ideas lies in the fact that this disorder sets up in the prostate nodular or diffuse indurated structures which, on clinical examination, may be mistaken for tuberculosis or a malignant tumour. Histological examination of the prostate may even confirm this incorrect diagnosis if one is unfamiliar with the histological picture of granulomatous prostatitis. Thus, before a diagnosis of tuberculosis of the prostate is given and the patient is subjected to prolonged antituberculosis treatment, before a diagnosis of cancer of the prostate is given and continuous treatment with œstrogens, castration or total prostatectomy is advised, one should think of ruling out non-specific granulomatous prostatitis, a benign disorder with a favourable prognosis.

Angina Pectoris and Hiatus Hernia.

Can hiatus hernia be regarded as one of the causes of anginal pain? P. Hillemand, P. Chiche and J.-M. Pernot[6]

[1] Verge, J., and Dhennin, L. and L. (1961), *Concours méd.*, 83:3611 (June 24).

[2] *Presse méd.*, 1961, 69:1324 (June 17).

[3] *Presse méd.*, 1961, 69:961 (May 6).

[4] *Sem. Hôp.*, 1961, 37:1908 (June 8).

[5] *Presse méd.*, 1961, 69:1392 (June 21).

[6] *Sem. Hôp.*, 1961, 37:1473 (May 2).

have attempted to elucidate this problem by investigating 100 patients with hiatus hernia. They were not concerned with discovering whether the hernia could cause pain resembling anginal pain, but with discovering whether an hiatus hernia could or could not set off anginal paroxysms, and whether this disorder could produce true angina pectoris or electrocardiographic changes that might lead to a diagnostic error between the two abnormalities. As a corollary to the question set, another arose—to discover whether it was possible to cure angina pectoris by treating an hiatus hernia. After discussing the statistical, clinical and experimental findings, the authors found that they had to abandon the implication of hiatus hernia as a cause of anginal pain. None of the clinical, statistical or experimental arguments seemed sufficiently convincing to permit the authors to state that an hiatus hernia alone had the capacity to set off attacks of true anginal pain. They finally considered that, in the absence of other discoverable cardiovascular causes, the occurrence of attacks of anginal pain retained its usual significance—coronary atherosclerosis.

Correspondence.

FLUORIDATION OF PUBLIC WATER SUPPLIES.

SIR: In response to your invitation in THE MEDICAL JOURNAL OF AUSTRALIA for October 7, may I, as an often misquoted opponent of fluoridation of public water supplies, express my opinion on the subject.

In 1939, following information then available, my children were already brushing their teeth with fluoride. I am not therefore, and never have been, opposed to the use of fluoride either internally or externally for dental purposes. I am, however, opposed on principle to the deliberate addition of any substance whatever to a public water supply with the avowed intention of influencing any physiological function of the human body. To yield on this principle will establish a serious precedent affecting the liberty of the citizen, and the responsibility of scientists in general and the medical profession in particular.

Chlorination of a water supply, be it noted, is done to destroy bacteria of disease, not to act upon the human body. The development of the teeth is a physiological function, and fluoridation of the water supply aims to influence that function. Alteration of physiological function is the object of all medication which, in fact, is so defined. Fluoridation of a water supply has as its object the mass medication of children up to the age of 11 years, the vital period of tooth development. It is deliberate sophistry to claim that fluoridation is not mass medication. The fact that people in certain regions consume water containing relatively high quantities of fluoride does not invalidate this argument. All the mineral constituents of water vary enormously, not only throughout the globe, but also in Australia. Just where does this so-called adjustment of the water constituents end? Why not increase the magnesium to alleviate widespread constipation?

Any medication, however simple, may in a given individual have unwanted or even harmful side effects. A physician is responsible under law to control medication of an individual patient in order to secure the most favourable and least undesirable effect. No two patients react alike. Mrs. Brown, who consults me, is not an item of statistics. I could not plead as a valid excuse in the coroner's court that I gave Mrs. Brown the average dose of a medicament and that I did not think it necessary to attend her to see how she reacted. Fluoridation of a water supply treats people in terms of statistics, not as individuals, of which no two are alike. Who assumes responsibility for a case of fluorosis resulting from deliberate addition of fluoride to a water supply?

There is a fundamental scientific basis for this responsibility of a physician. Any mixed population of living creatures demonstrates a wide variation in response to any biologically active substance administered internally or externally. The effect of alcohol on human beings is a highly contentious example. Some are so susceptible that they should never touch it, and others so constitutionally favoured as to be practically immune. Compulsory control by total prohibition of the use of alcohol has been rightly rejected by most societies. Compulsory consumption of fluoridated water is merely application of the same principle in reverse.

Variability in response is a universal law of living nature, and the arbitrary choice of a fluoride concentration of 1 p.p.m. in the water supply can neither prevent some sensitive individuals from developing fluorosis of the teeth, nor the insensitive children from failing to derive any benefit because the dose is inadequate. Individual administration and control of the clinical results by qualified personnel provide the only ethical and scientific safeguards. The dose of fluoride per individual depends on the amount of water consumed, and this, too, varies even more than the individual response of the teeth. Dental statistics are one thing, individual variations in reaction to fluoride and in the quantity of water consumed are quite another matter.

The cause of dental caries at this date is unknown. The manner in which fluoride inhibits caries is also unknown. There is a mass of published evidence that points to faulty nutrition as an important factor, but in what manner is also unproven. The use of fluoride is, therefore, empirical, not scientific. There is no doubt whatever that fluoride does reduce the incidence of dental caries. There are good scientific reasons for doubting the accuracy of the figures for percentage reduction that are so unblushingly quoted. Everyone realizes just how difficult it has been accurately to determine statistically whether smoking is a cause of lung cancer. It is unlikely the use of tobacco will ever be prohibited by the action of enthusiastic opponents.

For the purpose of argument, I will accept without prejudice a 50% reduction in total caries incidence, however estimated. This does not mean that 50% of children will develop no caries. It means that the total number of cavities in the teeth of all children is reduced by 50% compared with controls. Some will show little change, others a great improvement, but the figures refer only to averages. Approximately 5% will inevitably disclose varying degrees of dental fluorosis, which is a brown mottling disfigurement of the teeth. If by a remote chance your child proved to be a bad case, would the statistical explanation mollify you?

The major fluoride effect is produced only during the years of tooth development—that is, up to the age of 11 years. This age group comprises roughly 20% of the population. Therefore, for every 20 individuals for whom the fluoride is intended, 80 others, toothless or not, must imbibe the fluoride. Incredible though it seems, everybody must take the fluoride all their lives—that is almost seven times as long as is necessary to control the dental caries. This is therapeutically indefensible.

But this is not all. The Engineer-in-Chief, South Australian Engineering and Water Supply Department, has kindly given me his departmental estimates of water usage in Adelaide. I quote: domestic gardens, 50%; industry and commerce, 20%; primary production, 4%; drinking and kitchen use, 3%. Everyone knows just how large a part of that 3% is used for laundry, ablutions, wash-up and cleaning purposes. It would be a gross over-estimation to assume 1% for drinking, even if manufactured beverages were included. Therefore, speaking as a rate-payer—and this is a painful qualification—the economics of this proposal are an affront to my intelligence. At the very most, one-fifth of 1% of all this fluoridated water is actually effective for the purpose for which the fluoride is added. The rest goes on the garden, through the machinery and/or down the sewers.

When I ask my dental friends why they do not advocate the supply to and use by parents of fluoride tablets, and the control of the dental effect by the school dental service, I am invariably told that parents could not be relied on to cooperate. How do they know? I am unaware of any intensive campaign having been undertaken to advocate such a procedure in Australia. The general public could be pardoned for assuming that fluoridation is the only way to avoid caries. In fact, one dental proponent publicly stated that I was indifferent to the plight of the children, since I opposed fluoridation. Now I come to a reason which really aggravates my concern.

I attended an address to a recent Australian Dental Congress in Adelaide by a leading dental fluoridation expert of the United States Department of Health. He advised his listeners to press for fluoridation by influencing municipal councils and governments. He warned them not to permit the subject of fluoridation to become a matter for public debate because, he said, plebiscites were invariably against the proposal owing to the influence of crackpots.[1] In itself this is a remarkable tribute to the influence of

[1] The tide is running strongly against fluoridation in the United States of America today.

crackpots, and at the same time a contemptuous insult to the intelligence of the average citizen. It discloses, moreover, what in my opinion is a dangerous trend in our day and age. This is the tendency of the pseudo-scientific expert to use authority to impose his views. It is so in the United States where fluoridation is actively advocated with public funds by the dental experts of the United States Public Health Service, and it is also the case in New Zealand.

It is my conviction that if a medico-social measure cannot be sufficiently clearly explained to one's fellow men to win their confidence that it is honestly presented and that there is no other alternative to its adoption, there is something wrong somewhere. If we cease to base important social actions on argument with our fellow men and cease to accept each our individual share of responsibility—even in the matter of our children's teeth—we are merely proving that Krushchev's contempt for a free society is thoroughly deserved, and we may as well resign ourselves to being more than symbolically clubbed on the head with his shoe.

Yours, etc.,

C. STANTON HICKS.

Adelaide.
October 16, 1961.

COUNTRY DOCTOR. NO THANK YOU.

SIR: It is a little over 400 miles from Adelaide to Wudinna in the west of South Australia. Perhaps it is this reason which removes the attraction for enterprising doctors to entertain the idea of such country work.

For a number of years a Flying Medical Service with a high record of efficiency and enterprise has been sponsored by the Bush Church Aid Society. In 1947 a well-constructed hospital at Wudinna, South Australia, came under the Society's direction. Since that year doctors have served varying periods either sponsored by the Society or assisted by the local Council. Despite the widely advertised need, the District Council has been disappointed that a suitable doctor has not been forthcoming to relieve the anxiety of the people in this country area.

A strong plea has been made for the Bush Church Aid Society to relieve the situation. Here is a challenge for a qualified man or woman to join the Christian missionary staff. The well-equipped hospital has four double-certificated nurses and two hospital assistants, who are all women with a deep conviction of their Christian calling. A comfortable house is available and a pharmacy is to be established. Remuneration, travelling and practice expenses can be adequately covered.

I can but plead the hospitality of your columns to bring this need before the notice of a doctor who is looking for the opportunity to share in a really vital Christian missionary service in our own country. Surely there must be at least one doctor who will read this and take the trouble to let me have an inquiry.

Yours, etc.,

J. R. GREENWOOD,
Organizing Missioner.

Bush Church Aid Society,
B.C.A. House,
135 Bathurst Street,
Sydney.
October 26, 1961.

THE CONCENTRATION OF ADRENALINE IN LIGNOCAINE SOLUTIONS.

SIR: May we draw the attention of your readers to a potentially hazardous situation present in the adrenaline concentrations in "Xylocaine" brand of lignocaine?

This drug is packed in various strengths, with and without adrenaline. The commonest of these are: 1% "plain" (i.e., without adrenaline); 2% "plain"; 1% with adrenaline 1: 100,000; and 2% with adrenaline 1: 80,000. It is the last two, with adrenaline, which cause concern, for the following reasons.

1. With the concentrations of 1 in 80,000 and 1 in 100,000 adrenaline in these packs, only the smallest volumes (not more than 2 ml.) of a solution of this strength may be injected with safety. Even when diluted by an equal volume of water the concentration of adrenaline at 1:160,000 is still too high.

2. The label is printed "with epinephrine 1 in 80,000 or 1 in 100,000". This American version of adrenaline is not well known, and has led, in the experience of one of us, to the addition of further adrenaline, in the mistaken belief that the solution did not contain any! Fortunately this error was detected before administration.

3. Many doctors (wrongly) call for local anæsthetic agents with adrenaline, without bothering to check or specify the concentration of the latter. "Xylocaine" 2% with adrenaline is often offered and may cause serious reactions if injected rapidly in quantities in excess of 3 or 4 ml.

We believe that the offending pack was originally introduced for dental work, where a high concentration of adrenaline is called for, but correspondingly small volumes of solution are used. Now, however, "Xylocaine" has become the most commonly employed local anæsthetic in medical practice, and it is advertised freely in the pages of your Journal. We are of the opinion, therefore, that this totally unsuitable pack (for medical purposes) should no longer be sold to hospitals or doctors, and should be replaced by a more suitable solution, appropriately labelled.

In the meantime it is important that all practitioners using local anæsthetics should study the concentration of adrenaline in the solution of their choice, and insist that the concentration does not exceed 1 in 250,000.

Yours, etc.,

ROSS HOLLAND, F.F.A.R.A.C.S.,
PETER BLAXLAND, F.R.C.S.

Fairfield Hospital,
Fairfield,
New South Wales.
September 28, 1961.

SIR: The question about the optimum concentration of adrenaline in lignocaine solutions, brought up by R. Holland and P. Blaxland, is quite complicated. We would like to discuss just a few aspects which we consider to be particularly important.

Adrenaline is added to the solution in order to increase the efficiency (potency) of lignocaine, *i.e.*, to increase depth, spread and duration of anæsthesia, and also in order to decrease the risk for toxic reactions by slowing the rate of absorption from the injection site (by vasoconstriction). Lignocaine without adrenaline is quite rapidly absorbed from a local site and in highly vascularized areas, like terminal infiltration in the dental field, the addition of adrenaline is required for good anæsthetic effect. In this particular location the efficiency of lignocaine has been found to increase further if the adrenaline concentration is increased above 1:80,000, and this relationship has been found to apply also for nerve blocks in animals. We have evidences (to be published) that different local anæsthetic agents interfere to a varying degree with the local vasoconstrictor action of adrenaline, and since also the requirements for an optimal vasoconstriction vary in different locations, it is difficult to state which concentration of adrenaline should be considered to be the best from a practical point of view.

We certainly agree with Holland and Blaxland that it is important for the user to know the concentration of adrenaline in a particular solution. To avoid mistakes each concentration of lignocaine is therefore usually marketed only with one concentration of adrenaline. In spite of the extensive use of the different solutions all over the world for more than ten years there is no positive evidence that the amount of adrenaline chosen for these solutions is unsuitable. There is no reason, however, to use higher concentrations of adrenaline than necessary and clinical studies have shown that an adrenaline concentration of 1:200,000 is quite adequate for many medical applications. Since any desired adrenaline concentration can easily be obtained by mixing appropriate volumes of "plain Xylocaine" and "Xylocaine-epinephrine" a particular lignocaine solution with adrenaline 1:200,000 for limited purposes has been marketed only in a few countries. If there is a demand for such a solution of lignocaine among doctors in Australia we are sure that the Astra subsidiary there will be willing to make such a solution available to them. A change of the present labels should naturally also be considered to avoid mistakes in the future.

We are aware of the need in the medical field of a local anæsthetic agent which may be used without, or with low concentrations of, adrenaline and still possess adequate efficiency. Our research efforts in this direction seem to hold some promise. The introduction of a new agent with low adrenaline content for certain areas of application and

keeping the well-known "Xylocaine" solutions as they are may, from a practical point of view, be a better arrangement.

Yours, etc.,

AB ASTRA, S. WAHLQVIST.
Medical Department,
Sweden.
Undated.

COMMON INJURIES OF THE KNEE JOINT SUSTAINED IN AUSTRALIAN RULES FOOTBALL.

SIR: I feel the article by Dr. K. W. Hinrichsen, "Common Injuries of the Knee Joint Sustained in Australian Rules Football" is very timely. Sports injuries as a whole are usually fairly poorly managed and can lead to severe disability. Of particular importance, Dr. Hinrichsen's emphasis that a player must leave the field after even the most minor injury to the knee should be noted. This undoubtedly will cut down the eventual morbidity that occurs among athletes and sporting personnel.

Yours, etc.,

13 Colin Grove, G. M. BEDBROOK.
West Perth,
Western Australia.
October 17, 1961.

Notes and News.

Permanent International Festival of Medico-Surgical and Scientific Films.

The second session of the Permanent International Festival of Medico-Surgical and Scientific Films, organized by the Association Nationale des Médecins Cinéastes et des Cinéastes Scientifiques de France, in conjunction with the *Gazette médicale de France*, will begin on February 21, 1962, at the Faculty of Medicine, Paris. Producers of 16 mm. medico-surgical or scientific films, not released in France, who wish to take part in this film showing, should submit their entries no later than December 5, 1961. The Technical Committee appointed to judge the films will meet on that date. All entries must be addressed as follows: Association Nationale des Médecins Cinéastes et des Cinéastes Scientifiques de France, 23, Boulevard de Latour-Maubourg, Paris, VII°. Overseas entrants would be well advised to send their films to the Cultural Attaché of their Embassy in Paris, in the care of their Minister of External Affairs.

Ultra-Pure Water for Hospitals and Industry.

To meet the growing need in hospitals, manufacturing industries and general scientific research for purer water than can be obtained by multiple distillation, a British firm has introduced a range of low-priced portable water purifying units known as elgastats. It is stated that all the units will purify tap water without prior treatment and, dependent on size, will produce ultra-pure water at flows ranging from four to 50 gallons an hour. Independent of inlet water hardness, ultra-pure water of consistent quality can be drawn off until the capacity of the purifiers is exhausted. This latter stage is shown by a simple colour change or by a meter built into the apparatus. The basic unit, introduced commercially for the first time, is a simple cartridge containing a mixed bed of ion-exchange resins. These cartridges can either be recharged with fresh resins or replaced by a new cartridge, the exhausted material being sent back to the factory for regeneration. The company has set up a world-wide postal service. Although the standard cartridges reduce considerably the bacteria content of tap water, for absolute sterility it is necessary to irradiate purified water with ultra-violet light. This refinement can be built into the largest unit, a small movable cabinet, which gives an output of up to 50 gallons an hour, and can be used to provide ultra-pure water as an extra laboratory service.

New Building for Blue Cross Organizations in Sydney.

A new building to house the joint administration of the Medical Benefits Fund of Australia and the Hospitals Contribution Fund of N.S.W. has been erected at 258-262

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 21, 1961.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5(2)	2(2)	1(1)	..	..	..	..	..	8
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	2	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(1)	18(16)	6(2)	1	..	..	2	..	31
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	5	..	1(1)	16(4)	..	..	..	22
Encephalitis	1(1)	2(2)	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	181(82)	105(48)	19(11)	53(8)	3(2)	2(1)	..	7	370
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	2	..	..	..	2
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	4(4)	..	..	..	..	..	4
Meningococcal Infection	1	1	..	..	..	..	..	..	2
Ophthalmia	..	..	..	..	6	..	..	..	6
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	3	1	2	..	1(1)	..	..	..	7
Puerperal Fever	2(1)	..	..	..	..	..	..	..	2
Rubella	..	23(22)	..	1(1)	5(3)	2(2)	..	..	31
Salmonella Infection	..	..	..	1(1)	3(1)	..	..	..	4
Scarlet Fever	5(4)	7(4)	4(2)	6(5)	1(1)	2(1)	..	1	26
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	8(1)	..	..	..	8
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	40(32)	27(17)	11(6)	2(2)	12(8)	..	3	..	95
Typhoid Fever	1(1)	2(2)	..	..	..	..	..	..	3
Typhus (Flea-, Mite- and Tick-borne)	1	..	..	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

George Street, Sydney. It was officially opened on Saturday, October 7, 1961, by the Prime Minister of Australia, the Right Honourable R. G. Menzies, in the presence of a large and representative gathering which included a number of visitors from other States. The Prime Minister, who was introduced by the President of the Medical Benefits Fund of Australia, Sir Ronald Grieve, referred to the development of the medical and hospital benefits schemes. He emphasized the

fact that the medical benefits scheme maintained a middle-of-the-road course as compared with various approaches to the provision of medical care in other parts of the world. In particular, it preserved the precious doctor-patient relationship.

The new building, which is of 13 storeys, is fully occupied by the two funds, both of which are Blue Cross organizations. It brings together under one roof staff that has been scattered over six locations. This concentration of staff and the integration of the two funds which is now going on should greatly improve the efficiency and economy of administration of both funds.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Wark, Marian Lynette, M.B., B.S., 1959 (Univ. Sydney), 400 Darling Street, Balmain.

Seifert, Paul, M.D., 1947 (Univ. Heidelberg), registered under Section 17 (2) (*b*), *Medical Practitioners Act*, 1938 (as amended), 94 Old South Head Road, Bondi Junction.

Thompson, Patrick, M.B., B.S., 1960 (Univ. Sydney), 26 Scouller Street, Marrickville.

Deaths.

THE following death has been announced:

HEASLIP.—William Gordon Heaslip, on October 27, 1961, at Enfield, South Australia.

Diary for the Month.

NOVEMBER 13.—Victorian Branch, B.M.A.: Finance Sub-Committee.
NOVEMBER 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
NOVEMBER 16.—Victorian Branch, B.M.A.: Executive Meeting of Branch Council.
NOVEMBER 17.—New South Wales Branch, B.M.A.: Ethics Committee.
NOVEMBER 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
NOVEMBER 22.—Victorian Branch, B.M.A.: Branch Council Meeting.
NOVEMBER 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
NOVEMBER 24.—Queensland Branch, B.M.A.: Council Meeting.
NOVEMBER 28.—New South Wales Branch, B.M.A.: Hospitals Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): Medical Officers to Sydney City Council. All contract practice appointments in New South Wales. Members are requested to consult the Medical Secretary before undertaking practice in dwellings owned by the Housing Commission.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full data in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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